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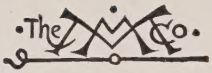
# CAVERNOUS SINUS THROMBOPHLEBITIS

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# CAVERNOUS SINUS THROMBOPHLEBITIS

AND

# ALLIED SEPTIC AND TRAUMATIC LESIONS OF THE BASAL VENOUS SINUSES

*A Clinical Study of Blood Stream Infection*

BY

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New York

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1926

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Set up and electrotyped.
Published July, 1926.

PRINTED IN THE UNITED STATES OF AMERICA BY
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TO MY MOTHER

IN LOVE AND VENERATION

WHO COMBINED "THE SPIRIT OF YOUTH

WITH THE WISDOM OF OLD AGE."

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## PREFACE

This monograph is the record of my personal experience. The cases are reviewed at length, and especial emphasis placed upon each and every diagnostic or operative error made; because, as I have learned chiefly by my failures, I have stressed them, so that all may profit by my experience. For I am persuaded that a study of the included cases will enable a diagnosis of thrombophlebitis of the cavernous sinus to be made early enough for rational surgery to be effectual, in a certain proportion of cases. In this way many lives may be rescued from a condition, which, up to the present, has almost uniformly terminated fatally.

I wish to extend thanks to my colleagues of the Newark Eye and Ear Infirmary and the Newark City Hospital for placing cases and records at my disposal.

Some of the material contained herein has been presented in part before the following societies:

(1) Philadelphia College of Physicians (Section of Otolaryngology), February 18, 1920.

(2) New York Otological Society, November 27, 1923.

(3) Chicago Otolaryngological Society, December 3, 1923.

(4) Philadelphia College of Physicians (Section of Otology and Laryngology), February 20, 1924.

(5) New York State Medical Society, Rochester, N. Y. (Section of Ophthalmology and Otolaryngology), April 23, 1924.

WELLS P. EAGLETON.

Newark, N. J., April, 1926.

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# CAVERNOUS SINUS THROMBOPHLEBITIS

# INTRODUCTION

A proper understanding of the intracranial complications of aural and nasal suppuration hinges on the mechanism of the venous or the return flow from the brain to the heart. Personal experience has taught that by far the larger proportion of all cases of brain abscess and meningitis are caused by the infective process traveling by way of an infective retrograde thrombophlebitis, and not by direct extension of the suppuration; this latter condition has become progressively rarer with the advent of early attack on the primary focus in the mastoid and accessory sinuses. During the past few years nearly all the cases of meningitis and brain abscess coming under my observation have been preceded by a well-conducted mastoid or nasal sinus operation, the subsequent operative or post-mortem findings demonstrating that the infection entered the dural envelope by a retrograde thrombophlebitis, either of a basal sinus or a small venous radicle. Consequently, a consideration of the suppurative lesions of the venous system must underlie our surgical treatment not only of sinus thrombophlebitis, but also of meningitis and brain abscess.

**Material.**—This contribution is primarily the study of 25 personally observed cases of cavernous sinus thrombophlebitis with 21 deaths (twelve autopsy reports) and four recoveries, three of which were

verified by operative finding (cases 12, 22 and 23) and one (case B) questionable; the clinical evidence not being sufficient to warrant a positive diagnosis that the cavernous sinus itself was involved. The outcome of one case (case 8) is unknown.

There also enter into the discussion a rather extensive experience with thrombophlebitis of the jugular bulb and experiences with cases of infective orbital thrombophlebitis—numerous cases of arteriovenous aneurism of the cavernous sinus, with recovery following operation; one case of infective thrombophlebitis of the lateral sinus and jugular vein on the side opposite to the primary aural suppuration, with autopsy-protocol; two cases of ascending lateral sinus thrombophlebitis from suppuration in neck; and two cases of longitudinal sinus thrombosis—all of which are of sufficient rarity to warrant their separate consideration.

**Object.**—The chief object of this study is to emphasize the importance of the following considerations in the diagnosis and treatment of infective cavernous sinus disease:

(1) If the diagnosis is to be made early—which it must be, if the surgical attack is to have the slightest chance of success—cavernous sinus phlebitis should be regarded as a group of diseases; the same as we to-day consider the different types of nephritis not as "Bright's" or kidney disease.

(2) Again, for early diagnosis it must be appreciated that the classical symptoms of exophthalmos, edema of the lids, and chemosis may or may not be present, depending on whether the sinus is suddenly

and completely obstructed by an acute septic process, or gradually obliterated by a compensatory thrombus.

(3) An early diagnosis in the cases of slow involvement without classical manifestations of exophthalmos (the most promising type for surgical intervention) necessitates a careful study of the following mechanical factors which determine the symptomatology:

(a) The path of entrance of the infection into the sinus;

(b) The part of the sinus first attacked; and

(c) The alterations in the adjacent tissue that occur as a result of the venous anastomosis of the sinus.

(4) The necessity of treating the phlebitis not only by drainage, but by placing the inflamed venous radicle at rest by ligation of the common or internal carotid artery.

So far as I am aware, many of the above considerations have not yet received the attention that their importance demands.

**Historical.**—The first knowledge of cavernous sinus thrombosis comes from Abercrombie,[1] who in 1818 described it from autopsy findings, and Duncan,[2] who in 1821 also observed it at a post-mortem. Clinically, Vigla [3] was probably the first to record it, in 1839; he was followed by Castleman and Ducrest [4] in 1846. Knapp,[5] in 1868, treats the subject along the lines of

[1] Abercrombie: *Diseases of the Brain,* 1818.

[2] Duncan: *Edinburgh Medical and Surgical Journal,* 1821.

[3] Vigla: *Thése de Paris,* 1839.

[4] Castleman and Ducrest: *Recherches sur les Abcés multiples,* 1846, p. 23.

[5] Knapp, H.: *Archiv. f. Ophthalmologie,* 1868, p. 220.

differential diagnosis very much as is done to-day; in this study he mentions Blandt, Dreyfuss, Gerber, Hajek and St. Clair Thomson. Dwight and Germain [6] in 1902, and Hartley [7] in the same year were the first to attack the sinus surgically.

**Frequency.**—Dwight and Germain [8] in 1902 collected 178 cases from the literature. Smith,[9] in 1918, estimated that less than 300 cases had been recorded. Chisholm and Watkins [10] found but eight cases in 50,000 surgical records of the Johns Hopkins Hospital.

However, it seems that reliable statistics are not as yet available; for, in my own experience, I have found the disease is certainly not so infrequent if carefully sought for in septicemia and pyemia during life, or at autopsy following general blood stream infections. This contention is supported by a review of the literature, which suggests that a diagnosis of cavernous sinus thrombophlebitis is rarely made in the absence of exophthalmos and ocular chemosis; though the latter is far from infrequent. And the cavernous sinus is seldom examined at autopsy, even in cases of sepsis, unless other venous sinuses have been involved. Hence, many cases are overlooked.

In two of the cases herein reported, a thrombophlebitis of the cavernous sinus would have passed unnoticed had

[6] Dwight, E. N., and Germain, Harry: "Thrombosis of the Cavernous Sinus," with report of four cases, including one cranial operation, *Boston Medical and Surgical Journal*, 1902, p. 146.

[7] Hartley and Knapp: *Archiv. of Ophthalmology*, Vol. 29, 1900.

[8] Dwight and Germain: *Boston Medical and Surgical Journal*, May, 1902.

[9] Smith, D.: "Cavernous Sinus Thrombosis," with notes of five cases, *Archiv. of Ophthalmology*, 47:482, September, 1918.

[10] Chisholm and Watkins: *Arch. Surg.*, November, 1920.

not a careful search been made at the post-mortem. The apparent focus of sepsis in one patient was a chronic disease of the antrum (Case 3), and the other patient had suffered from sphenoidal suppuration (Case 16).

The realization that a septic clot may fill the cavernous sinus of both sides—the only symptom present being high, continuous temperature or chronic sepsis, has necessitated a review of all of the writer's cases, with the formulation of certain deductions which, it is hoped, may lead to a better understanding of the subject.

**Longitudinal Sinus Phlebitis.**—In a similar way, of the two cases of superior longitudinal sinus thrombosis, one had outstanding symptoms of great disturbance to the return circulation,[11] while the other was an accidental post-mortem finding, the patient dying of meningitis without clinical or laboratory evidence of vascular obstruction or blood stream infection.[12]

[11] Eagleton, Wells P.: *Brain Abscess. Its Surgical Pathology and Operative Technic.* Macmillan, 1922, p. 129. Case 23.
[12] Eagleton: *Loc. cit.*, p. 71. Case 15.

# Cavernous Sinus Thrombophlebitis

## CHAPTER I

### ACUTE AND CHRONIC TYPES—ROUTE OF INFECTION

**Importance of Distinguishing Clinically Between Sudden and Gradual Obliteration of the Cavernous Sinus.**—Clinically, cavernous sinus thrombophlebitis presents two distinct and separate types—one acute, the other a chronic septic condition; the first giving rise to outstanding diagnostic symptoms of *sudden* obliteration of a large venous trunk from an acute and complete venous blockage of the veins emptying into the cavernous sinus; the second presenting no symptoms of venous engorgement, because a slow occlusion of the sinus has allowed time for the venous blood to establish other channels. Consequently, it is necessary that these two types of cavernous sinus thrombophlebitis be clinically differentiated if the surgeon is to determine whether the classical localizing symptoms (high temperature, chills, sudden exophthalmos, chemosis, first of one eye, then of the other) will or will not be present at an early stage.

I. **Acute infection of the cavernous sinus,** *from extension of a thrombophlebitis of the veins which empty* into the sinus anteriorly—the ophthalmics—

associated with a sudden occlusion of the lumen of the sinus.

These are the cases described in all the text-books, the classical symptomatology of chemosis and exophthalmos being caused by the purulent thrombophlebitis reaching the sinus by way of the ophthalmic veins. The infection is in the eyelid, or the ethmoidal cells, traveling through the pterygoid plexus from the teeth, or by the facial and angular veins, from the lip or orifice of the nose.

This type (acute obliterating septic thrombophlebitis of the cavernous sinus by extension of infection from the veins emptying into its anterior portion) is the most common, not only because of the proximity of the inflammation, but also because all the venous radicles of this region have few valves, which not only allow a retrograde thrombophlebitis, but, by permitting a reversal of the venous current (with the resulting "ebb and flow" movement of the blood) are favorable to the formation of a thrombus.

Acute anterior cases present little difficulty in diagnosis; they are early accompanied by chemosis, exophthalmos, first of one eye and then of the other, associated with profound pyemia and followed by death from sepsis or a complicating meningitis.

Case 1. (Italian Boy.)

*Fulminating Cavernous Sinus Thrombophlebitis from Ulcerated Tooth.*

*History:* Twelve days previously had ulcerated tooth with pain and swelling of jaw and face. Dentist extracted two teeth. Followed by bilateral exophthalmos and chemosis with high temperature. Death from acute sepsis.

II. Slowly Obliterating Thrombophlebitis.—Thrombotic occlusion of the cavernous sinus may, however, occur without localizing symptoms, as the production of exophthalmos and chemosis requires a sudden disturbance of the return venous circulation before the collateral circulation has time for readjustment. Consequently, when the cavernous sinus is slowly obliterated, or when the part of the sinus first involved does not suddenly interfere with the return ophthalmic flow, then the classical symptoms of chemosis and exophthalmos may not be present at all; or will be of such late appearance as to be useless for early diagnosis.

Case 2.

*Chronic (Compensatory) Cavernous Sinus Thrombophlebitis of Aural Origin Without the Classical Localizing Symptoms Until Fifty-second Day of Disease.*

*Previous history* of chronic catarrh of nose and throat; and bilateral chronic otitis.

*November 18:* Sudden right-sided earache and dizziness. Vomited two days later. Earache persisted but dizziness disappeared.

*November 27* (nine days after first complaint of earache): Patient had a chill lasting one-half hour. Both ears now discharging profusely; no pain.

*December 1:* Temperature reached 104° F. and continued high for five days and then became normal. Following few days had several chills.

*December 6* (eighteenth day): Patient was up and about, although he had a chill that night, followed by fever.

*December 14* (four weeks after original earache): Developed double vision. On the fourth day of double vision, went to physician's office; said he "felt well"; but

complained of headaches and pains in his muscles. *Said nothing about ear.*

*Diagnosis:* "Paralysis of the external rectus of right eye of unknown origin."

*January 8:* Wife reported that although patient was up and about, attending to his work and felt fairly well, he had chills and fever at night and would go to sleep at any time during the day from "progressive weakness." A few days later, first complained of severe pain in neck.

*January 9* (fifty-second day of disease): Patient now looked severely ill (cachectic); talked through nose; said he had difficulty in swallowing for past three days and in addition to discharging ears and paralysis of external rectus, had a large fluctuating mass in right nasopharynx.

*Examination:* (Drs. Theo. Corwin and Henry Barkhorn.) "Bilateral complete paralysis of sixth, partial paralysis of right third, bilateral choked disc. Right mastoid tender; swollen gland under right mastoid, almost complete destruction of both drums; hears with both ears." Temperature, 100° F.; pulse, 96. On account of bilateral paralysis of the sixth and a partial paralysis of the right third, with a mass in the nasopharynx, a diagnosis of empyema of the sphenoid pointing into the nasopharynx was made. Intra-oral incision of abscess liberated more than an ounce of foul-smelling pus, and bare bone was palpated, extending upward to the base of the skull; examination of the pus showed a mixed infection—pneumococcic.

Four days later, temperature again rose. Both sphenoids explored through normal opening. From the right a gelatinous cystic mass was removed, followed by about fifteen drops of seropurulent fluid.

*January 13: First appearance of slight exophthalmos and chemosis (fifty-sixth day of disease and thirtieth day after the invasion of the cavernous sinus as shown by the ocular paralysis), optic neuritis worse, with hemorrhage into retina. Continuous septic temperature.*

*January 15:* Sphenoid and ethmoid sinuses exenterated

under cocaine; mucous membrane found to be hyperplastic. Exodus (fifty-ninth day of disease).

*Autopsy:* Pus and organized clot in right lateral sinus, which extended into the transverse sinus of the opposite side to within an inch of unaffected (left) mastoid. Free pus in right jugular vein continuous with that in the jugular bulb and lateral sinus. Free pus and old organized clot in right inferior petrosal sinus; free pus in entire cavernous sinus on both sides. Right superior petrosal could not be found. Pus in sphenoidal sinuses.

*Remarks:* The importance of the case centers (1) in the slow, almost afebrile but progressive character of the infective thrombophlebitis, which (unsuspected during life) had extended from the jugular bulb to the cavernous sinus, and then *retrograded through the pterygoid plexus* into the pharynx, causing a retropharyngeal abscess; (2) in the absence of any signs of exophthalmos or chemosis until very late in the disease, because of the slow development of the thrombosis; (3) in the intimate relationship between pre-existing chronic sphenoidal sinus disease and an intercurrent infection in the causation of cavernous sinus thrombophlebitis.

In view of the latency of the chronic type, if the surgeon is to diagnose its presence early enough to be of real surgical assistance, he must analyze all cases: (a) according to the route that the infective process has taken to enter the sinus, and (b) according to the different pathological processes present. For in the chronic compensation type a diagnosis at times can be accomplished only by abstract reasoning, as in many cases its sole basis will be the possibility that an extension of the suppurative process through its communicating venous channels into the cavernous sinus *might* produce the symptoms present in the suspected case.

A. **Classification Based on the Route of Invasion Into the Sinus.**—Cavernous sinus thrombophlebitis may be divided into six groups, depending upon the *route* by which the sinus is originally infected.

(1) *Ophthalmic* (*anterior*), the infection entering the anterior part of the cavernous sinus from in front by way of the ophthalmic vein. Following an infection, generally staphylococcic, of the lip, eyelid, nasal orifice, teeth, or the anterior part of the mouth, there develops a retrograde thrombophlebitis which ascends through the angular or facial venous plexus, enters the orbit and is transmitted by the ophthalmic veins to the cavernous sinus. This is also the usual route of invasion of the ethmoid cases that perforate the orbit.

(2) *Pterygoid* (*anterior-inferior*), the infection attacking the sinus in its forward and lower region. The infection here reaches the sinus from below by way of the pterygoid plexus, as it ascends in the pterygoid fossa with its numerous anastomoses from the pharyngeal plexus, through the foramen ovale, the deep facial, and the inferior ophthalmic veins. These cases usually originate from foci in the posterior teeth, jaw or maxillary antrum, less frequently in the pharynx or the tonsils. When cavernous sinus phlebitis originates from the tonsil, the path of the invasion is generally indirect, involving first the internal jugular vein and from it ascending into the inferior petrosal. (See Group 5.) However, cases are on record in which the cavernous sinus thrombophlebitis extended directly from the tonsil into the pterygoid plexus, with which the tonsillar veins anastomose.

(3) *Sphenoidal* (*mesial*), the infection attacking the mesial portion of the sinus by (a) caries of the sphenoid or from (b) a thrombosis of the veins which pass through the sphenoidal roof into the cavernous sinus.

(4) *Aural* (*posterior*), the infection entering the posterior portion of the sinus (a) from thrombosis of the *inferior petrosal,* or (b) from caries of the deep cells at the apex of the petrous pyramid, where it impinges on the body of the sphenoid and unites with the petrous of the opposite side. (Case F.)

(5) *Tonsillar* (*posterior-inferior*) *infection through the pterygomaxillary fossa, by way of an ascending thrombophlebitis of the jugular vein.* In these cases the cavernous sinus is first attacked in its posterior and lower portion by way of the petrosal sinus. Following a peritonsillar abscess or operation on the tonsil, a thrombophlebitis of the tonsillar veins passes through the superior constrictor muscles, giving rise to a thrombophlebitis of the jugular vein, from which it retrogrades into the jugular bulb. Here, because of the venous "eddies" and the obliquity of the entrance of the inferior petrosal into the jugular bulb, the septic process easily involves the inferior petrosal, through which it reaches the cavernous sinus.

(6) *Carotid venous plexus with direct infection into the lumen of the cavernous sinus*—a microscopical pathway; in which a jugular bulb infection (from either the ear or the throat) extends through the internal carotid canal in the skull, and thus first reaches the anterior portion of the cavernous sinus. Throm-

bophlebitis of the venous plexus surrounding the internal carotid artery, stands clinically between the aural (posterior) and the ophthalmic (anterior), as, although of aural origin, it early gives rise to exophthalmos.

**Mixed Types.**—All six types, although anatomically and clinically distinct, may pass from one to another because of the free anastomotic communication of the venous radicles with each other. Thus a pterygoid plexus (anterior-inferior) infection frequently first attacks the sinus from in front by way of the ophthalmic veins, thus becoming orbital. (Case 7.) A tonsillar infection may extend either by way of the pterygoid plexus, or through the jugular vein and inferior petrosal. The posterior (aural) may become medial (sphenoidal). (Cases 2 and 20.)

Each of the above six types may or may not present the classical localizing symptoms of cavernous sinus thrombosis, *viz.*, exophthalmos and chemosis, depending upon whether or not the circulation in the cavernous sinus is suddenly and completely occluded.

## RÔLE OF SUPERINFECTION IN THE CAUSATION OF CAVERNOUS SINUS THROMBOSIS

It is a clinical fact that cavernous sinus phlebitis, apparently of aural origin, is often associated with a sphenoidal suppuration. Multiple areas of infection are frequently present during life and at post-mortem; it may be impossible to decide which was the primary cause of the thrombophlebitis (Cases 2, 20, and F).

A study of the cases herein reported would suggest that in several the cavernous sinus thrombophlebitis

was precipitated by an intercurrent disease, such as acute tonsillitis or acute otitis media, which awakened a dormant sphenoidal sinus suppuration. The cavernous sinus phlebitis, while possibly not the direct result of the chronic sphenoidal disease, probably would never have developed but for the superinfection (tonsillitis or otitis) and its associated "depression immunity." (Morgenroth.[1] [2] [3]) Cases 2, 20, and F, all had acute aural and chronic sphenoidal disease.

This superimposed element I believe to be of considerable clinical and diagnostic value; although its importance apparently has not been recognized.[4]

B. **Classification Based Upon the Pathological Condition of the Sinus. The Rôle Played by Thrombus Itself.**—From a surgical standpoint, cavernous sinus cases must be further divided according to the virulence of the pathological process present in the sinus, into (1) Acute (Fulminating) and (2) Chronic (Compensatory).

A surgical attack upon the sinus in the acute fulminating cases can hold out little prospect of success because of the severity of the infection, unless the

[1] Morgenroth, J., Biberstein, H., and Schnitzer, R.: *Die Depressions-immunitat, Deutsche med. Wchnschr.,* 46:337, March 25, 1920.

[2] Teale: *Loc. cit.*

[3] Jessop's case: had tonsillitis and sphenoid.

Jacques and Lucien's case: had peritonsillar abscess, and osteomyelitis of sphenoid and petrous.

Whitehead's case: had tonsillitis and sphenoid.

O'Malley's case: followed adenoid operation.

[4] Eagleton, Wells P.: *Operative Treatment of Suppurative Meningitis.* Case 3 of fulminating meningitis from recrudescence, by exposure and vaccine treatment. Tr. American Otological Society, 1921.

operative procedure be early instituted and radically executed. On the other hand, the chronic (compensatory) cases offer a moderate hope of the patient's recovery from surgical intervention because of the slow development of the occlusion and the largely protective nature of the clot. The surgical attack, however, must be undertaken before the meningeal spaces have been invaded by the purulent process.

Case 3.

*Acute Fulminating Cavernous Sinus Thrombophlebitis from Acute Pharyngitis or Chronic Antrum Suppuration.*

(Death in three days from meningitis and sepsis,—no ocular symptoms. Autopsy.)

*History:* Brought to the Newark City Hospital in a semi-comatose condition. The previous evening the patient had applied for a room in a hotel and asked for a drug store to obtain something for a "terrible" headache; and in the morning was found in a stuporous condition. On admission to hospital "man looks acutely ill; talks little and wants to be left alone; puts hand to front of head, complaining of intense pain on being disturbed." He was sufficiently aroused, however, to make the statement that he had had a severe headache for the last day and a half, and that several days ago he had an operation on his nose. In his pocket was found a card from a clinic in a neighboring city, which stated that a tooth had been removed to drain the right antrum, and that it had been irrigated several times.

*Examination:* There is an oval drainage hole over the canine tooth of the right side, partially draining the antrum. Tonsils and pharynx, red. Left frontal sinus slightly tender to pressure and seems denser on trans-

illumination. Marked rigidity of neck. Reflexes, normal. Temperature, 103° to 105° F. Pulse, 100 to 130. Ophthalmoscopic examination: margins of both discs distinct, veins slightly enlarged. Lumbar puncture, purulent fluid from which direct smears showed numerous streptococci, gram positive, and diplococci, no intracellular organisms.

Died on second day in deep coma with profound sepsis.

*Post-mortem* by Dr. Harrison S. Martland.

*Oropharynx:* The entire pharynx and larynx are of a deep scarlet red color, also the mucosa of both nostrils, the latter being bathed in a considerable thick, purulent secretion. A probe can be passed into the right antrum through the right nostril. The antrum is free and clear although the mucous membrane is greatly thickened; the old drainage wound through its alveolar process is almost closed.

*Accessory Sinuses:* The right frontal sinus is practically obliterated; the left is free from suppuration. The ethmoids and sphenoid are free from infection.

*Cranial Cavity:* On removing the dura the subarachnoid space of the cortex shows a purulent greenish yellow exudate scattered generally over both cerebral hemispheres; and reaches the median line. This exudate is also present at the base (general suppurative meningitis). The vessels at the base are quite normal except the cavernous sinuses —both of which contain a recent reddish, grumous thrombus. The left cavernous sinus contains free pus; a greenish purulent exudate also surrounds it. There is no edema of the retro-orbital tissue and no protrusion of the eyes.

*Probable Cause of Death:* (1) Acute inflammation of naso-pharynx as manifested by extreme injection of mucosa of nose and upper respiratory tract. (2) Suppurative meningitis, due to venous septic thrombosis of cavernous sinuses following infection from naso-pharynx. (3) Acute cloudy swelling of heart, muscle, and liver. (4) Toxic nephritis. (5) Acute splenitis. (6) Hypostasis of lungs.

RELATIVE FREQUENCY OF THE DIFFERENT SITES OF THE PRIMARY INFECTION RESULTING IN CAVERNOUS SINUS THROMBOSIS OF THE 25 CASES HEREIN REPORTED.[5]

Acute fulminating, 17 cases (1, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 16, 18, 19, 20, 21 and 22)—68 per cent.

Chronic compensatory, 8 cases (2, 13, 14, 15, 17, 23, 24 and 25)—32 per cent.

Anterior invasions, 13 cases (1, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 21 and 22)—52 per cent.

Posterior invasions, 7 cases (2, 13, 14, 15, 17, 20 and 23)—28 per cent.

Mesial invasions, 4 cases (16, 18, 19 and 25)—16 per cent.

Internal invasion (carotid venous plexus), 1 case (24)—4 per cent.

| | *No. of cases* | *Per cent.* |
|---|---|---|
| From infections of face, eyelids, nasal orifice, lips and teeth. | 10 | 40 |

| | *No. of cases* |
|---|---|
| Infection from wound of face—Case 4 | 1 |
| Infection of eyelids—Cases 11 and 12 | 2 |
| Infection of nasal orifice—Cases 5 and 8 | 2 |

[5] From an analysis of 140 cases, Smith found that infection originated from the

| | |
|---|---|
| Ear in | 40 per cent. |
| Orbit and face in | 35 per cent. |
| Mouth and throat in | 13 per cent. |
| Nose in | 9 per cent. |
| Other causes in | 3 per cent. |
| | 100 per cent. |

Smith, Dorland: "Cavernous Sinus Thrombosis," with report of five cases, *Arch. of Ophth.*, 1918, Vol. 47, p. 482.

| | *No. of cases* | *No. of cases* | *Per cent.* |
|---|---|---|---|
| Infection of teeth—Cases 1 and 7 | 2 | | |
| Infection of lips—Cases 6, 10 and 21 | 3 | | |
| From antrum and ethmoids | | 4 | 16 |
| Infection of antrum—Cases 3 and 19 | 2 | | |
| Infection of ethmoids—Cases 9 and 22 | 2 | | |
| One from antrum disease following tooth (Case 7). | | | |
| One from acute sinus disease (Case 22). | | | |
| From gland in neck—Case 17 | | 1 | 4 |
| From sphenoid [6]—Cases 16, 18 and 25 | | 3 | 12 |
| In which sphenoid played a part, Cases 2, 13, 16, 18, 20 and 25. | | | |
| From ear—Cases 2, 13, 14, 15, 20, 23 and 24 | | 7 | 28 |
| Two had sphenoid disease sufficient to cause symptoms associated with suppurating ear (Cases 2 and 20). | | | |
| | | 25 | 100 |
| From the carotid venous plexus following a jugular bulb thrombophlebitis, Case 24 | 1 | | |
| In which underlying leukemia was chief factor, Case 19 | 1 | | |
| In which trauma played a part, Cases 3, 4, 5, 6, 10, 12, 17, 18 and 19 | 9 | | |
| Associated with embolic vascular occlusion, Case 11 | 1 | | |

[6] O'Malley (referred to in Dorland Smith) says, "It is a remarkable fact that several of the cases that have followed operations on the tonsils have been found to have caries of the sphenoid."

# CHAPTER II

## ACUTE OPHTHALMIC OR ANTERIOR INFECTION

### A. ACUTE (FULMINATING) THROMBOPHLEBITIS CAUSING AN OBSTRUCTION IN THE ANTERIOR PART OF THE SINUS FROM INVASION OF THE OPHTHALMIC VEINS.

(a) **From Wounds and Infections of the Face, Teeth and Eyelids.**—To this group belong the cases that are described in all textbooks; they present early outstanding diagnostic symptoms as the result of the sudden venous obliteration and are characterized by high temperature, chills, chemosis, exophthalmos, first of the eye of the homolateral side and then of the opposite eye, with partial recession of the proptosis of the first eye and rapid death from sepsis or meningitis. The swelling of the face so frequently seen is occasioned by a thrombophlebitis of the facial veins. (Coupland.) (Case 6.)

CASE 4.

*Acute Cavernous Sinus from Thrombophlebitis of Angular Vein.*

*History: August 1,* was struck at root of nose by a cricket ball, followed by great swelling of the left eye and left temporal region. Three days later the right eye began to swell. Following day discharge of pus from left lid; "since when swelling of left side of face has greatly subsided."

*August 6:* Admitted to Newark Eye and Ear Infirmary (service of Dr. Fred'k Jacobson). Patient feels "weak and dopey" but can be aroused. Temperature 97° F.

*Examination:* Slight edema on a line from the bridge of nose to right frontal eminence. Pus flowing from sinus at lower outer edge of left lid. Exophthalmos of both eyes; ocular movements limited, but right eye not entirely fixed. Left fundus: retina pale and edematous. Veins full; arteries small. Right, cannot see arteries in fundus. Vision: left, no perception nor projection; right, recognizes fingers.

*Operation:* Counter opening made through skin in front of left ear.

*August 7:* Right eye moveable by pressure quite freely. Can move upper lid volitionally. Sight unchanged. Temperature from 103° to 105° F.

*Death:* (7 days from injury).

(b) **Operative Trauma: A Cause of Precipitating a Staphylococcic Blood Stream Invasion from Infections About the Face and Lips.**—In staphylococcic infections (furuncles and carbuncles) of the face, especially of the lips, operative incisions in the early stages not only frequently fail to reveal pus in the furuncle but have been followed by a rapid extension of the inflammatory area.[1] It is now known that staphylococcic local infection involves the small veins,[2] but this is circumscribed—local thrombophlebitis—at an early stage before the formation of an abscess; if, however, the local area is now incised, and especially

[1] Gallenaerts: "Furuncle of the Face and Orbital Thrombophlebitis," *Medécine,* Paris, January, 1923, Vol. 4, No. 4, p. 255. Abs. J. A. M. A., 1923, advocates that every furuncle of the face should be treated early and radically by the galvanocautery.

[2] Bothe, Albert E.: "Staphylococcus Aureus Septicemia," *The Medical Clinics of North America,* January, 1923, Vol. 6, pp. 1029-1052.

if it is further traumatized by squeezing, the bacteria are disseminated. See illustration after page 138. The surgeon thus converts a local process into a general blood stream infection, which through a retrograde thrombophlebitis of the angular and facial veins, causes cavernous sinus involvement and death.

CASE 5.

*Acute Fulminating Cavernous Thrombosis of staphylococcic origin, with a complicating hemolytic streptococcic blood stream involvement, from pimple of nose and operative trauma. Autopsy.*

*Previous History:* Generally good. Compound fracture of the leg thirteen months previously, and "has not felt well since."

*Present Illness:* Two weeks before admission to hospital a small pimple appeared inside the right nares, which was opened by a physician. Immediately the nose and surrounding tissues and right eye became swollen and red. Again incised at bridge of nose, following which inflammation spread to scalp.

On the sixth day of the disease, temperature was 102° F. and patient delirious. No pain in the head. Two days previously "sore spots" appeared on the calf of the right leg, the deltoid region of right arm and on both ankles (acute sepsis). Admitted to Newark City Hospital with diagnosis of facial erysipelas.

*Examination:* Patient acutely ill. Face purplish red and swollen, with an irregular line of demarcation which spread to the occiput. Over the bridge of the nose are yellow papules exuding thin yellow fluid. Small dried areas of subsiding inflammation over the left external malar region. No pain in head.

*Eyes:* The lids of both eyes are closed by edema. Marked chemosis and bilateral exophthalmos.

*Blood:* Leucocytes 38,000, polynuclears 93 per cent.,

lymphocytes 7 per cent. Blood culture: staphylococcus aureus.

*December 21:* Patient irrational, delirious and semicomatose. Temperature, 102° to 106° F. Second blood culture: staphylococcus aureus and streptococcus hemolyticus. Pulse, 90-154. Respiration, 28-44.

*December 22:* Death (on twelfth day of invasion).

*Autopsy:* Both cavernous sinuses filled with pus from retrograde thrombophlebitis of the pterygoid plexus. Purulent infiltration surrounding the veins entering the sinus from in front. Cellulitis of face. Septicemia.

Lip infections often cause thrombophlebitis of the angular vein which passes through the facial and causes jugular thrombosis without retrograding upward into the sinus. (Case 6.)[3] [4]

Case 6.

*Thrombophlebitis of facial veins with general blood stream infection (probably from internal jugular) originating from pimple of lip.*

*History: July 8,* Small "pimple" on right side of upper lip. Opened by needle; that night her lip gave her so much pain that a physician was called, who treated it locally for 6 days.

*July 15:* Admitted to infirmary.

*Examination:* (Dr. John V. Bissett.) "The whole right half of upper lip indurated and swollen, closing the right eye. About half an inch above the vermilion border is a pus point, and half an inch below the vermilion border

[3] Reverden autopsy report abstracted by Martin, W., in *Annals of Surgery,* Vol. LXXVI, No. 1, p. 15, July, 1922.

[4] Martin, Walter: "Staphylococcic Infections of the Face and Lip;" *Annals of Surgery,* Vol. LXXVI, No. 1, p. 13, July, 1922, reports seven cases of lip infections with death in six and two autopsy reports, one revealing cavernous sinus phlebitis and the other no thrombophlebitis but septic *infarcts* in the lungs, etc. There is good bibliography of lip infections.

another pus point. Edema of lids of left eye. Severe pain. Very little temperature (99° to 100° F.).

*Operation:* (J. V. B.) Ethylchloride; "connected the two suppurating points. *No free pus* but numerous pin points of pus. The *tissue* was hepatized both in color and consistency. Very little bleeding."

*July 16:* The face and right eyelids less swollen, can open eye. Fundus, negative.

*July 18:* Conscious and rational but did not rest well. Temperature, 104° to 105° F.; pulse, 82; respiration, 22. Blood: erythrocytes 4,600,000, leucocytes 84,000; polynuclears 89 per cent., small lymphocytes 8 per cent.

*Operation (2nd):* "Radiating incisions made throughout buccal surface of right side of lip and face; raised the tissue from the malar bone. Tissue hepatized." Pus from lip; streptococcus, staphylococcus, and gram + bacillus.

*July 19:* Temperature 105° F. Leucocytes 10,200. Polynuclears 84 per cent., small lymphocytes 5 per cent., large lymphocytes 7 per cent., large mononuclears 4 per cent. Blood culture: staphylococcus albus. Urine: hyaline and many gram + casts.

*Operation (3rd):* Several more incisions, opening lip through and through. Blood transfusion. Following this, severe chills, and patient became delirious.

*July 21:* Unconscious. Death 13 days after infection.

In five of my own cases operative trauma certainly played the exciting part (Cases 6, 10, 12, 19 and 21); while in two others, although the acute symptoms followed a surgical manipulation, the disease itself probably was present at the time.

(c) **From Antrum Diseases by invasion of the anterior portion of the cavernous sinus through the ophthalmics.**—When the infective process originates in the teeth, it may occasion an acute osteomyelitis of the superior maxilla, with involvement of the antrum;

this rapidly perforates the orbit and extends to the cavernous sinus. In these cases the infection travels, not by continuity of tissue suppuration, but by retrograde venous thrombosis, which latter is the cause of the nutritional death of the osseous parts.

The accompanying acute osteomyelitis is the result of the venous infection—not the cause of it.[5] In such a case drainage of the associated orbital abscesses can not possibly control the disease, as the infection has already invaded the sinus itself.

The literature shows the uniformity with which futile drainage operations on the orbit and maxillary antrum and ethmoids have been performed—futile from failure to attack the thrombophlebitic nature of the process itself.

These cases, if diagnosed early and dealt with by surgery to the thrombotic veins, should offer a fair prospect of recovery.

CASE 7.

*Acute cavernous sinus phlebitis with osteomyelitis of antrum from a tooth infection. Autopsy.*

*History: April 3.* Came to the clinic of the Newark Eye and Ear Infirmary with an extensive suppuration of a right upper molar tooth and an infiltration of the eyelids and face. Tooth ordered drawn.

*April 8:* Admitted to infirmary (service of Dr. E. A. Sherman) with abscess of right orbit; temperature, 103° F. Abscess incised. During the night became restless and voided involuntarily. Had slight cough. Temperature, 101° to 105° F. Pulse, 128. Respiration, 54 to 60. Chest, negative.

[5] Charlin's Case 3. *Ann. d' Oculistique,* November, 1920.

*April 9:* Enlarged veins in both conjunctivæ; apparently a slight exophthalmos of both eyes. Fundus veins in right very full. Vision: right 20/200, left 20/50.

*April 12:* Swelling over temporal, parotid, and zygomatic regions, with enlarged veins. Veins of the bulbar conjunctiva less marked than two days ago. Cyanosis over left eyelid. Today right eyeball has apparently returned to normal position; left has exophthalmos. Fundus margin of right disc slightly blurred; left normal. Pupils equal. Death in deep coma twelve days from beginning of infection.

*Post-mortem:* (Dr. F. A. Sutton.) Removing the roof of the right orbit revealed free pus at the apex but outside the orbital tissue itself; which latter contained a gangrenous area connected with a perforation in the floor of the orbit which entered the right antrum. The sphenoid wing of the orbit was also infiltrated. The sphenoidal sinus was absent (as is usual in the case of young children), in its place there being a fine cellular structure which was not involved. The tissues of the left orbit were edematous. The posterior ethmoid cells of the right side were also involved. Exposure of the floor of the middle fossa disclosed a number of dilated veins on the right side, which were not present on the left side. The right cavernous sinus contained free pus, which extended through the circular sinus. The left cavernous sinus also contained free pus.

*Comment:* It would appear from the history that the infection had originated from a tooth, from which a retrograde thrombophlebitis ascended upwards, causing bony necrosis of antrum, and ethmoids, and orbital abscess from nutritional death of tissue, involving the cavernous sinus, first on the right side and then through the circular sinus to the left side.

(d) From tonsillar suppuration by way of the pterygoid plexus, associated with an acute obstruc-

**tion of the anterior portion of the sinus.**—These almost invariably have an acute thrombophlebitis of the ophthalmic veins empting into the sinus, which in itself gives rise to an orbital abscess and frequently disguises the involvement of the sinus until the infection has extended through the circular sinus to the opposite side.

### ORIGIN OF SYMPTOMS IN ACUTE ANTERIOR CASES

The symptoms present may be divided into those due to:

(1) *Venous obstruction* to the return circulation: Exophthalmos, chemosis, local edema, dilated veins.

(2) *General septic manifestations* from the blood stream infection: Temperature, chills, malaise, sweating, metastatic abscess.

### CLASSICAL SIGNS

*Causes of chemosis and swelling of the lids.*—Chemosis may be caused by an edema following disturbance of the return venous circulation; but this of itself is seldom the sole cause in cavernous sinus thrombophlebitis. To produce a severe chemosis, suppuration must be added, either an inflammatory infiltration of the orbital tissue surrounding the thrombosed veins (Case 11), or an orbital abscess. The severe edema of the eyelids so frequently seen in cavernous sinus phlebitis is not entirely due to thrombophlebitis of the ophthalmic veins.[6] Most of the swelling of the lids is a lymphedema from blocking of

[6] Coupland: *Tran. Ophthal. Soc. of United Kingdom*, 1888, p. 240.

the lymph channels by inflammatory exudations as lymph vessels are numerous in the lids and conjunctiva but of small importance in the orbit.

In the presence of chemosis an effort should be made to differentiate between inflammatory lymphatic edema and that from venous stasis. The chemosis of venous origin results from transudation into the tissues from excess of venous blood supply, while the lymphatic vessels of the lids and conjunctiva are not affected. Clinically, chemosis from venous stasis is associated with dilatation of the anastomotic veins, which leave the orbit anteriorly by way of the angular vein. The eyelids are of a bluish hue, while individual dilated veins can frequently be seen under the skin. The mobility of the eyeball, although interfered with by the associated exophthalmos, is not completely lost as in orbital abscess. This is the clinical picture presented by arteriovenous aneurisms of the cavernous sinus from fracture of the skull. (See illustrations, Cases H and I.)

In chemosis of infective origin, the lymphatics are plugged by exudate and are thus no longer able to function.

Of course, in invasion of the cavernous sinus from in front (ophthalmic cases), there are both venous obstruction from the thrombosis and infection of the tissues in the neighborhood of the infected ophthalmic veins. Here the chemosis and associated exophthalmos are chiefly orbital. But in the chemosis and exophthalmos that originate from the sinus itself (and the latter are very rare), differentiation between lymphatic and venous obstructive origin should be possible.

CASE 8.

*Phlebitis Resembling Orbital Abscess.*

*History:* Sore inside of left nostril a few days before admission to hospital, at which time (although the original infection had healed) the patient gave all the appearance of a left-sided orbital abscess; *viz.*, exophthalmos, chemosis, swelling of lid and blindness of left eye. However, the *chemosis was too slight for the degree of exophthalmos, and the veins of the ocular conjunctiva, lid and surrounding tissue were tortuous and distended.*

Patient slightly stuporous and apathetic, with general septic appearance; temperature, 101° F.; no chills.

*Operation:* "Enucleation of eye and evisceration of orbit revealed infiltrated mass from a thrombophlebitis process in the orbit. No pus." Result unknown.

*Causes of Exophthalmos.*—McCallum and Cornell [7] have demonstrated experimentally that exophthalmos may be caused by venous engorgement alone, but the venous obstruction must be sudden and more or less complete. This condition is far from infrequent, and clinical diagnosis may be possible by exophthalmos in the absence of chemosis, as in the following case.

CASE A.

*Non-suppurative thrombophlebitis of ophthalmic vein from ethmoiditis or infected teeth, manifested by exophthalmos without chemosis. Spontaneous recovery.*

Patient of Dr. E. A. Curtis.

*Previous History:* Patient had toothache on right side about a year ago; none since. Subject to frequent colds in head, with chronic expectoration.

[7] MacCallum, William C., and Cornell, B.: "On the Mechanism of Exophthalmos," *Medical News,* October 15, 1904, p. 732.

*Present Illness:* Five days previously complained of pain over right frontal region and on top of head. Right eye became swollen, followed by double vision.

*Examination:* Pronounced exophthalmos of the *right eye without* chemosis but associated with diplopia. Pupil in moderate dilatation reacts to light and accommodation. Fundus, dilatation of blood vessels. Tenderness over right frontal sinus. X-ray shows both antrum and ethmoid cloudy. Unextracted roots, with positive signs of infection. Temperature, 102° F. Blood count: leucocytes 21,000, polynuclears 87 per cent., lymphocytes 13 per cent., hemoglobin 85 per cent., erythrocytes 3,650,000. Wassermann, negative. Blood culture, negative. Urine, negative. Rapid subsidence of all symptoms.

*Diagnosis:* Non-suppurative phlebitis of ophthalmic blood vessels of right orbit from infection of antrum of Highmore and ethmoids.

Post-mortems show that the venous engorgement is rarely the sole cause of the exophthalmos in cavernous sinus phlebitis, as with chemosis the exophthalmos is largely due to an inflammatory infiltration which surrounds the thrombosed veins in the orbit. Moreover, there is frequently an associated orbital abscess, even in those cases in which the ophthalmic vein has not carried the infection into the sinus, the ophthalmic veins being involved from the sinus phlebitis itself.[8] (Case 3.) In such a case, the orbital abscess is a symptom of perforation of the outer wall of a venous radicle (ophthalmic) and not of obstruction to venous flow. The surgical lesson is that exophthalmos should

[8] Turner: Case: from tooth, four days' duration, gave no signs of thrombosis on left side during life, but post-mortem showed septic thrombosis of both cavernous sinuses, and the orbital veins of the right side were thrombosed, with an orbital cellulitis—the cause of the exophthalmos. *Lancet,* May, 1900, p. 1467.

not routinely be regarded as either of orbital or of venous obstructive type, but, if the cavernous sinus is suspected, the surgeon should explore the orbit early, and failing to locate an abscess, should radically attack the sinus itself.

Case B.

*Thrombophlebitis of veins of orbit with resulting orbital abscess—osteoplastic flap. Recovery.*

*History:* Three weeks previously had pain in tooth in left upper jaw. For past week complains of pain and swelling of left eye and left side of face.

*October 27, 1919:* Admitted to infirmary. Dr. Wm. Brien.

*Examination:* Swelling of outer portion left side of face. Exophthalmos, left. Eyeball immovable. Ophthalmoscopic examination negative, except dilated veins at periphery. Vision, L. S., see movement of hand at 6 inches. R. S. 20/20. Temperature, 101.4° to 103° F. Leucocytes 16,000, polynuclears 72 per cent., large lymphocytes 16 per cent., small lymphocytes 12 per cent. (Wasserman negative.)

*Diagnosis:* Orbital cellulitis and frontal sinusitis, with secondary abscess.

*October 29, Operation:* Circular incision two-thirds around left orbit. Krönlein osteoplastic flap through the outer margin of the orbit, liberated pus from orbit; separate collection of pus found beneath the zygomatic arch extending to the root of the first bicuspid tooth and entering the left antrum. (Staphylococcus.)

Uneventful recovery.

*Comment:* While not a case of cavernous sinus thrombophlebitis, the accessory veins of the cavernous sinus were undoubtedly involved, as it cannot be otherwise explained how the pus got into the orbit.[9] It illustrates, furthermore,

[9] Chisholm and Watkins, Case 1. Infection by way of pterygoid plexus from tooth, secondary abscess in temporal region.

that in septic condition of the orbit a large osteoplastic exposure is the proper procedure.

In the chronic compensatory type of sphenoidal or aural origin, exophthalmos is rarely present at an early stage; chiefly because the infection first attacks the posterior portion or the body of the cavernous sinus; but, in addition, because a low type of infection occasions a gradual obliteration of the sinus so that no symptoms of venous engorgement result; for with a slow obliteration the return circulation from the eyes has ample time to create new anastomotic channels. The exophthalmos and chemosis when present are slight; and, as previously stated, are generally due to the inflammatory process secondarily invading the ophthalmic radicles by a retrograde thrombophlebitis (Cases 2 and 20), after the cavernous sinus has become involved.[10]

It is important that the surgeon carefully distinguish between a slight proptosis and an apparent one from restricted ocular movements which accompanies the paralysis of the 3rd, 4th and 6th nerves. The latter have been frequently mistaken for exophthalmos.[11]

### DURATION OF ANTERIOR CASES

The majority of the anterior cases are more or less acute in their course; the infective process generally lasting from 10 to 20 days before the fatal termination. Valuable time is usually lost in treating the infection

[10] Knapp (Arnold), in *Medical Ophthalmology*, states that 72 per cent. of all cases have exophthalmos. He evidently includes all cases, even when the exophthalmos is a terminal manifestation.

[11] Coupland: *Loc. cit.*

as if it were a suppuration limited to the apex of the orbit instead of a phlebitic process. However, fulminating cases are far from infrequent, death resulting in one of my cases 12 hours after the appearance of symptoms of thrombotic occlusion of retinal vessel (Case 11), and in another case, 4 days after an ethmoid involvement and 10 days from the beginning of the coryza (Case 9).[12] [13]

Case 9.

*Fulminating Cavernous Sinus Thrombophlebitis from coryza (ethmoiditis or sphenoiditis?)*

Patient of Dr. Frederick J. Wort.

Had a cold in head—thought to be ordinary coryza, but for several days had severe pain in nasal sinuses. Suddenly bilateral exophthalmos and chemosis, general swelling of nose and face—high temperature, cyanosis. Death on following day.

### COMPLICATING MYOCARDITIS FROM ACUTE ANTERIOR CASES

Cavernous sinus thrombosis from staphylococcus frequently causes valvular vegetations.[14] One of my cases had an acute endocarditis from the blood stream infection which had already caused a destructive change in the heart, but was not appreciated prior to operation. (Case 10.)

[12] Chisholm and Watkins: *Loc. cit.* In Case 11 death occurred twenty-one hours after the patient walked into the hospital, and five days after the initial "boil" on his nose.

[13] Hurd, L.: "Cavernous Sinus Thrombosis from Folliculitis of Nares." Staphylococcus. Died in five days. *Laryngoscope,* November, 1921, Vol. 31, p. 882.

[14] Whitehead: *Jour. Laryngology and Otology,* April, 1904, p. 181.

Case 10.

*Acute obliterating thrombophlebitis of the cavernous sinus and the ophthalmic veins from pimple of lip and operation. Death during operative trauma from hemorrhage and septic endocarditis.*

*History:* Nine days previously a pimple on the lower lip; this was opened by the patient's mother. The next day lip was very painful, and swelling increased, temperature 103° F. On 6th day a physician freely incised lip evacuating only a small amount of pus. The operation was followed by exophthalmos of the left eye on the succeeding day. Eighth day, right eye became swollen; patient very restless, complained of severe pain over left eye, failing vision, and pain in the neck (the general muscular pains of sepsis).

*Condition on admission* to infirmary on the 9th day of infection. Patient wildly delirious, with stiff neck which has been present off and on for past few days. Continuously high temperature and very rapid pulse (septic endocarditis). Diffuse swelling of face with suppurating wound of lip from which pus exudes (staphylococcus); bilateral bluish swelling of eyelids—chemosis and exophthalmos, chiefly left. (Typical picture of Cavernous Sinus Thrombophlebitis.) Lumbar puncture: globulin not increased; cell count low, 4 per cm.; reduces Fehling's solution; sterile.

*Operation:* Patient cyanosed from beginning (Mosher's operation); evisceration of orbital contents which liberated a few drops of pus from apex of orbit (staphylococcus). Sphenoidal fissure located without difficulty, through which emerged thrombosed enlarged veins. Sinus exposed by removal of overhanging great wing of sphenoid externally and below to floor of middle fossa. Dura separated from floor of fossa; could see difference in appearance between dura externally when it covered brain and internally where sinus was located. Opening of sinus failed to cause bleeding but no real clot found.

Sudden death during manipulation from septic endocarditis, and loss of blood during operation, plus ether.

**Septic Venous Emboli.**—From the nature of the process, dislodgement of septic emboli must frequently occur in thrombophlebitis. It is a common terminal manifestation, giving rise to infective infarcts in the kidneys, spleen, and lungs, while the presence of multiple small abscesses in the liver is a frequent cause of jaundice.[15]

There is some evidence that it is the dislodgement of septic particles that causes the repeated chills in cases of pyemia; however, in thrombophlebitis of the cranial sinuses septic venous emboli seldom involve a terminal vessel of sufficient caliber to give localizing manifestations, although this is far from uncommon in non-infective thrombosis. (See Case G.)

The following is the only case of cavernous sinus origin with post-mortem that has been reported, so far as I know.

Case 11.

*Fulminating cavernous sinus thrombophlebitis, from stye on eyelid, with slight symptoms until an embolic occlusion of ophthalmic vessel twelve hours before death. Post-mortem.*

*History:* Always subject to headaches—has had many styes—one week ago stye on right eye treated by his physician (Dr. Goudy).

*April 28:* Went to shop but complained on returning, "must have caught cold as muscles of neck were thick and

[15] MacCuen-Smith, S. B.: "Middle-ear Disease in Its Relation to Metastatic Abscess of the Liver and Other Viscera," *Archives of Otology,* Vol. XXXIII, 1904, No. 2, p. 87.

had difficulty in swallowing." Stye on right eyelid; left slightly swollen and sore.

*April 29:* Temperature, 101° F. Stye doing well.

*April 30:* Seen by physician about 5 p. m. Patient stated that all day he had been having very violent pain in left side of head, did not complain of left eye, and the swelling of lid was so slight as to escape casual attention. Two hours later, suddenly lost sight of left eye. Pain in head very much worse.

*Examination:* 8:30 p. m. Patient rational and readily got out of bed for examination.

*Eyes:* Bilateral exophthalmos slight of right, more marked of left, which latter has some chemosis of lower portion of conjunctiva. Left eyeball tender on pressure, pupil moderate size. Cornea beginning to be hazy but can make ophthalmoscopic examination, which shows general whiteness of fundus as if blood supply was absent; white disc but no atrophy.[16] Region beyond disc has marked dilatation of one of the veins. Fundus of right eye normal. Auscultation over left eyelid gives normal vascular bruit but less distinct than over right eye.

*Throat:* Chronically diseased tonsil on left side.

*Heart:* Indistinct murmur.

Two hours after examination patient unable to walk because of stiffness of muscles; had to be carried on a stretcher into hospital; temperature, 105° F. Delirious; rapidly became unconscious. Death, twelve hours after development of embolic blindness, and seven days from the beginning of the original infection.

*Post-mortem:* (Dr. Harrison S. Martland.) Skin over dependent portions of body cyanosed, finger tips blue. No redness or edema of tissues around the eyes or over bridge of nose.

*On removing the calvarium,* the dura over cerebral hemispheres tense but clear. Outer surface of hemispheres smooth and flattened, such as seen in increased

[16] Kolb's case.

intra-cranial pressure; moderate dilatation of vessels of pia.

*On removing brain,* small hemorrhage about the left carotid artery, also considerable hemorrhage around pituitary gland; area of free hemorrhage one by two cm. in diameter, at base of brain in the pia and arachnoid in the region of the left anterior clinoid process. No meningitis.

*Basal sinuses:* Left pterygoid vein markedly distended (being about five to ten mm. in diameter); contains large dark red clot easily removed, leaving clean intima. Right pterygoid vein small and normal.

Left cavernous sinus contains an irregular dark red granulated clot filling entire cavity; from the anterior part of this pus can be expressed. This clot extends from the left cavernous sinus through the posterior and anterior circular sinuses over into the right cavernous sinus, where the clot is a great deal smaller than on the left side. Sinuses of the posterior fossa normal.

*Orbits:* Left orbital cavity shows considerable edema and hemorrhagic infiltration of muscles and retrobulbar tissue behind eye. Same condition on right side but much less marked.

*Nasal Sinuses:* Sphenoidal sinuses show small blood clot extending to floor; both, however, are free from pus. Ethmoid cells normal. Both mastoids and middle ears normal.

*Body:* Cloudy swelling of heart. Atheroma of aorta. Lungs show small hemorrhagic infarcts. Spleen: large, soft and full of blood. Kidneys, normal size; parenchyma, cloudy and slightly swollen.

*Comment:* It would seem from the clinical history and the autopsy that the infection of the left cavernous sinus originated either from (1) a stye of the eyelid; (2) from the teeth or tonsil, or (3) possibly from the sphenoid. It is certain that for six days the patient had a low grade of sepsis which suddenly became fulminating, causing multi-

ple hemorrhage into the pia-arachnoid of the brain and occlusion of the ophthalmic artery or vein. (Pial hemorrhage occurs in acute vascular infection from streptococcus.)[17]

[17] Eagleton, Wells P.: *N. Y. Otol. Soc.*, January, 1917; *Ann. Otol. Rhino. and Laryngol.*, 1917, Vol. 26, p. 1060.

# CHAPTER III

## ANTERIOR CHRONIC (COMPENSATORY) CASES

### B. ANTERIOR CHRONIC (COMPENSATORY) PTERYTOID PLEXUS AND ORBITAL CASES

These, although much less frequent than the acute fulminating type, do occur.[1] [2] (Case 12.)

CASE 12.

*Acute followed by chronic blood stream infection from trauma to a staphylococcic "pimple" on eyelid giving rise to thrombophlebitis of orbital veins with cavernous sinus involvement. Treatment—multiple operations and serum injections. Recovery.*

Patient of Dr. A. Strausser.

*History:* One week previously the patient had succession of styes; last one on right lid which healed in three days; immediately after was accidentally struck by a man's elbow over site of stye, followed by tenderness, but not

[1] Chisholm and Watkins: Case 4, the patient lived over a month after the appearance of eye symptoms; the post-mortem demonstrated that the infection had extended from a tooth into the cavernous sinus, from which it spread into the orbit, causing a secondary orbital abscess; it later invaded the lateral sinus and the jugular.

Chisholm and Watkins: Case 8, the patient was ill over two months. Staphylococci thrombophlebitis from injury to nose, with recovery. The eye symptoms disappeared after three weeks, but recurred on the opposite side three weeks later, and were transient, lasting only two days.

[2] Talko: *Z. f. Augenhlk.*, 1900, Vol. IV, p. 236. Cavernous sinus disease from frontal sinus and orbital phlebitis lasted three months; second eye not involved for sixty days.

much swelling. Three days later considerable swelling of lid but little pain. Eyeball apparently normal.

*June 28:* (Fourth day after blow.) Upper lid increased in size. Eyeball limited in motion, but no exophthalmos or chemosis. O. E. normal. Patient extremely ill; temperature, 104° to 105° F. Septic erythematous rash over dorsal surface of legs and thighs.

*June 29:* Swelling of lid greatly increased but still very little pain; beginning right exophthalmos and chemosis, fundus, normal. Temperature continuously high, 105° to 106° F. Stock antitoxin serum (P. D. & C.) administered.

*June 30:* Today patient complains of pain at inner corner of eye. Exophthalmos and chemosis so great it is hard to keep lid covered. Beginning steaminess of cornea making fundus examination impossible. Slight indistinctness of margin disc of opposite (right) nerve head.

*July 2:* Patient very seriously ill. Irregular delirium. Bilateral deafness (septic); drum membrane normal. Temperature, 104° to 106° F. Respiration, labored. Pulse, 120.

*Operation:* Multiple exploratory incisions of orbit. *No pus located.* Injection of anti-streptococcus serum repeated.

*July 3-4:* Patient thoroughly septic; rational when awake; slight cough. Septic diarrhoea. Beginning metastasis in various parts of body (swelling over first metacarpal unassociated with pain; right knee boggy, left knee red), and erythema of skin. Heart has a soft murmur with first sound. Veins of lower part of thorax enlarged. Incision of superficial abscess on upper lid evacuated pus (staphylococcus albus).

*July 6-9:* Patient's life despaired of; intermittent delirium and restlessness; constant cough. Pain in head and abdomen. Temperature, 104° to 106° F. Pulse, 140.

*July 10:* Decided change for better; skin moist, but patient still delirious and has headache. Markedly deaf in both ears, although drum membranes are normal. Swelling of lids subsiding. Left cornea completely ulcerated. Papilledema of opposite fundus (right) increasing.

*Subsequent History:* Irregular temperature for 60 days,

100° to 103° F. Intermittent pain in head, face and temporal region. Considerable vomiting. Papilledema of right disc increasing steadily, then gradually subsided.

*Treatment:* Eyeball enucleated. Repeated injections of serum. Several metastatic abscesses incised and one sequestrum removed from dorsal surface of hand.

Recovery.

## CHAPTER IV

### AURAL OR POSTERIOR CASES WITH OR WITHOUT PROPTOSIS

Cases of cavernous sinus thrombosis of otitic origin may originate from (1) an adjacent suppurating process in the apex of the petrous pyramid (acute osteomyelitis). Here the blood stream in the sinus is suddenly and completely blocked; in which case classical symptoms are apt to be present. These latter cases are rare.[1]

The aural cases are more commonly due to (2) a gradual retrograde extension into the cavernous sinus of an already existing thrombophlebitis in one of the petrosals. As in the former case, if the vascular occlusion is sudden and complete, there will be localizing symptoms—if gradual, none will be present.

Consequently, for the purpose of early diagnosis, cavernous sinus and thrombophlebitis of otitic origin should be divided into:

(a) *Petrous pyramid* group from direct extension.

(b) *Petrosal sinus* group *from preëxisting blood stream infection,* by a retrograde thrombophlebitis. Of course, both groups are frequently associated with bony caries of the deep cells, which may give rise to a (c) *primary petrosal sinus disease.*

[1] Ballance, Blake and Charlin's cases.

(a) **Petrous Pyramid Group from Involvement of the Cells of the Cancellous Tissue at the Apex of the Petrous Pyramid.**—Girard [2] has described five groups of cells which surround the labyrinth, all of which tend to coalesce at the apex of the petrous pyramid.

Involvement of one of the perilabyrinthine cells—which is apt to be overlooked at the primary mastoid operations because of the interposition of the labyrinth—usually gives rise to a localized meningitis in the floor of the middle fossa. Rarely, however, one of the apical cells actually impinges on the cavernous sinus and may thus occasion a primary thrombus within its lumen.

These cases are almost invariably of the slow (compensatory) type, with vague symptoms of chronic sepsis—irregular chills, feeling of malaise, headache, and night sweats—and if recognized early and properly treated should give a fair prospect of recovery.[3] They are rare.

Gradenigo's syndrome—of running ear, mastoid involvement and sixth nerve paralysis—in the presence of acute or chronic sepsis, should awaken the suspicion that the sinus may be involved.[4] Of course, in Gradenigo's syndrome, the paralysis is much more frequently the result of an inflammatory "pinching" of the sixth nerve as it passes from the posterior to

[2] Girard, L.: "Perilabyrinthine Cells," *Int. Zentralbl. f. Ohrenheilk.*, 1912, exp. 156.

[3] Bircher: To this group belong Bircher's case, *Zentralbl. für Chir.*, 1893, p. 483, this being the only case of aural origin on record of recovery after operation on the sinus itself.

[4] Gradenigo, G.: "Ueber die Paralyse des Nervus abducens bei Otitis," *Archiv für Ohrenheilk*, B. 72-74, pp. 149-187.

the middle fossa over the apex of the petrous bone and under the sphenopetrosal ligament.[5] [6] [7]

CASE 13.

*Unsuspected chronic obliterating cavernous sinus thrombophlebitis of aural origin of four weeks' duration associated with chronic meningitis, presenting no localizing symptoms until two days prior to death, when ocular paralysis developed, followed by exophthalmos on day of death.*

*History of Illness:* "Grippe" five weeks previously (January 17), cough, coryza—headache, general pains; associated with several chills, and fever for a few days; accompanied by painful right ear with discharge, which latter has continued irregularly ever since. Following week, left ear became painful, which also discharged for about a week.

While patient has been in bed off and on since "grippe," she was much better until two days before admission; although she complained of cough, irregular headache on top of head, pains in left side of neck, fever at night and general malaise.

*February 21:* Severe pain in head and neck, and slight delirium: high temperature, rapid respiration. During day developed drooping of left lid and slight protrusion of ball. Examination by Dr. Charles Teeter: "Semi-conscious but can be aroused." Ptosis of the left lid, marked dilatation of the left pupil and fixation of the eyeball.

[5] Mosgrove, J. R.: Case of Septic Thrombosis, both ophthalmic veins and cavernous sinuses, *Ohio State Medical Journal,* August, 1913. Infection from nose—few days' duration, classical symptoms —no pus—death. No post-mortem.

[6] Ellett: "Septic Thrombosis of the Cavernous Sinus," *J. A. M. A.,* 1904, Vol. 43, p. 865. Incision revealed "no pus."

[7] Perkins, Charles E.: "Abducens Paralysis and Otitis Media Purulenta," *Ann. Otol. Rhino. and Laryngol.,* Vol. XIX, pp. 692-704, 1910; an analysis of 101 cases of Gradenigo's syndrome. Gives illustration showing relationship of motor oculi nerves with superior and inferior petrosal sinuses.

Left pupil does not respond to light. No rigidity of neck; no Kernig. Temperature, 102° F. Pulse, 100. Respiration, 30.

*Chest:* Crepitant and subcrepitant rales at both bases.

*Tentative Diagnosis:* Influenza, otitis media (possibly mastoiditis), cavernous sinus thrombosis.

*February 22:* Admitted to the infirmary in deep coma. Rigid neck; Kernig; delirious. Blood culture, negative. Lumbar puncture: yellowish fluid; pressure tremendously increased; Fehling's not reduced; globulin + + + +, cells + + + +; lymphocytes 15 per cent.; polynuclears 85 per cent.; smear, streptococcus; cultures, streptococcus (non-hemolytic).

*Diagnosis:* Acute streptococcic leptomeningitis with cavernous sinus thrombophlebitis.

*Death:* Twelve hours after admission.

*Post-mortem:*

*Basal Sinuses:* Free pus (streptococcus mucosus) in the left lateral sinus extending from the jugular bulb to the torcular Herophili, which is occluded by clot. From the bulb the pus extended through the left inferior petrosal to the cavernous sinus of the same side; from which it passed through the anterior and posterior circular sinus to the right cavernous sinus, which was filled with clot. This latter portion was apparently aseptic. The left superior petrosal was free from infection.

*Mastoid and Accessory Sinuses:* The mastoid cells did not contain a large amount of pus, but deep in the *apical cells of the petrous pyramid toward the internal carotid artery, there was thick yellow pus of an old process.*

Sphenoid sinuses had a thickened membrane, but no pus.

*Meninges:* Cortex of brain showed isolated patches of pus. The basal cisterna was filled with a solid exudate. The exudate was chiefly in the chiasmal space, but also in the interpeduncular, and also involved the pontine medullary space. The cerebellar tonsils were denuded of meninges, with small hemorrhages of the cortex. The tonsillar regions of both cerebellar hemispheres appeared necrosed,

the arachnoid apparently being elevated from the cerebellum. (The process in the meninges looked like an old one.)

*Comment:* Chronic thrombophlebitis with a very low grade of complicating meningitis (largely protective), involving the basal cisterna; the meningitis suddenly became general. It was not until the cavernous sinus thrombophlebitis had extended well forward that the exophthalmos and chemosis (which appeared only the day before death) developed.

## SYNOPSIS OF BIBLIOGRAPHY OF AURAL CASES —NOT PETROSAL

Ballance.—Tr. 9th Internat. Otolog. Congress, Aug., 1912, 315.

Case 4.—Boy. Suppuration right mastoid region. One week's illness. Temp., 103° F.; pulse, 104. Proptosis right eyeball, and ocular paralysis. Right retinal veins full, and fundus normal.

*Autopsy:* The infection evidently originated from an acute osteomyelitis of the cells of the apex of the petrous. Petrous necrosed. Dura and tegmen adherent. Pus in right and left cavernous sinuses. No thrombosis of sigmoid or jugular vein. Basilar meningitis.

Braun.—Annals Otol. Rhinol. Laryngol., June, 1914.

Ear. Chronic suppuration. Post-mortem.

Charlin, C.—Ann. d. Ocul., Nov., 1920, abst. in International Med. & Surg. Survey, Feb., 1921, No. 8a-53. Sec. 8-46.

Chronic otitis, right; headache and fever for ten days, then exophthalmos left, and next day of right.

*Post-mortem:* Basal meningitis in anterior part of base, and then orbital abscess.

Davis, H. J.—Proc. Roy. Soc. Med. (3), 1912-1913.

Davis.—Cerebellar abscess; cavernous sinus thrombosis. Proc. Roy. Soc. Med., 1919-1920, 13/3, 68.

Ear. Lateral sinus infection. Had a cerebellar abscess.

Delsaux.—Jour. Laryngol., Vol. 19, 1904, p. 484.
Ear, short duration. Autopsy.

Dwight and Germain.—Boston Med. and Surg. Jour., May, 1902.
Case 1: Ear. Five weeks. Autopsy.
Case 2: Carbuncle of the neck. 2 days. Autopsy.

Freisner, Isidore.—Simultaneous bilateral cavernous sinus thrombosis twelve hours after a simple mastoid operation. Ann. Otol. Rhinol. Laryngol., June, 1914, pp. 410, 440. Ear 8 days.

Gallaher, T. J.—Thrombosis of the intracranial venous sinuses of otitic origin.
Col. Med. Jour., April, 1909.

Goldthwait, R. H.—Thrombosis of the cavernous sinus, of otitic origin.
Mil. Surg. Wash., 1921, XLIX:430.
Suppuration followed right radical mastoid operation at inner end of petrous portion of temporal bone, extended along the line of the carotid canal to operative field.
*Autopsy:* Brain slightly enlarged. Ventricles contained considerable increase of fluid. At base yellowish pus followed along the course of meningeal veins. Right lateral sinus thrombosed, about 2 cm. on either side of surgical injury. Right cavernous sinus contained old thrombus.

Hanna, Henry.—"Fatal case of cavernous sinus thrombosis following chronic purulent otitis."
Jour. Laryngol. Rhinol. Otol., London, July, 1908.
Neglected suppuration of left ear. Finally acute, ending in death on 23rd day.
*Autopsy* showed dura mater corresponding to gap in roof of middle ear thickened and infiltrated with pus, but diseased area limited to size of a shilling. Extreme edge of left lateral hemisphere of cerebellum showed slight area of diseased and adherent dura mater.

Henning.—Wash. Med. Annals, Vol. 15, p. 241.
General infection direct from the ear; six months' duration; classical symptoms.

HORN, HENRY.—"Cavernous sinus thrombosis due to organisms of the streptothrix group." Ann. Otol. Rhinol. Laryng., Dec., 1913.
Ear. 22 days. Autopsy.

HUTCHINSON, JON., JR.—Tr. Ophthal. Soc. of U. K., Vol. VII, p. 250.
General infection. Death.

LANGWORTHY, H. G.—"Thrombosis of the cavernous sinuses, with report of four cases."
Boston Med. and Surg. Jour., April 25, 1907.
Anatomic relation of the cavernous sinus to the other structures.
Ann. Otol. Rhinol. Laryngol., Vol. 25, pp. 554-586.
Records cases not reported, by Sudler, M. T. Geiger, Jacob, Gifford, Colt, Lynch, Kylee, Scholz.

LOMBARD.—Abstracted by Ballance from Trans. 9th Int. Otol. Congress, 1912; 315.
R. otitis, herpes around mouth and nose. R. eyelid swollen on 16th day.
*Autopsy:* Revealed infected thrombosis of R. cavernous sinus. Suppurative meningitis. No visible tract from ear.

MAYBAUM, J. L.—"Cavernous sinus thrombosis of otitic origin."
Ann. Otol. Rhin. Laryngol., 1921, 30:1061.

MACEWEN.—Diseases of the brain and spinal cord. Ear. 30 days. Post-mortem.

MERCIER, BELLEVUE.—"Thrombophlébite suppureé des sinus caverneux d'origine otitique sans thrombophlébite du sinus latèral." Revue hebdom. de laryngol., etc., I, 1904, p. 401.

MOSHER, HARRIS B.—The Laryngoscope, Aug., 1914, p. 709.
Lateral sinus infection. Few days' duration. Death.

NEUMAN, D. S.—"Case of thrombosis of cavernous sinus following mastoiditis."
Col. Med. Jour., 1909, p. 548.

Raoult and Pillemont.—Bull. de Laryngol. Otol. Rhinol., Vol. 8, 1905, p. 107.
Ear. 2 months. Post-mortem.

Rodger, T. Ritchie.—Jour. Laryngol., 1921, p. 171.
Case 3. Acute. Death 5 days.

Robinson and Hutchinson.—"Case of ophthalmoplegia." Tr. Ophth. Soc. of U. K., Vol. VII, 1887, p. 250.
Pain in right temple and behind right ear; pain in right eyeball; slight exophthalmos.
*Autopsy:* R. temporal lobe apex softened, adherent to wall of fossa, and to sinus. Sinus obliterated by connective tissue and marked thickening of carotid artery. 3rd nerve swollen. (Good plates showing normal and diseased cavernous sinus with gradual obliteration giving rise to few symptoms.)

Thomson, St. Clair.—"The causes and symptoms of thrombosis of the cavernous sinus." Ophthalmic Review, Vol. XXVII.
In discussion of tonsillar cases Davis mentions one ear case of few days' duration.

Yersin.—Phlebite infectueuse du sinus caverneux.
Rev. Méd. de la Suisse Rom., Vol. 26, 1906, p. 456.

(b) **Cavernous Sinus Secondary to Retrograde Thrombophlebitis of One of the Petrosal Sinuses.**—These are the usual cases of aural origin—the infection originating from an aural suppuration, which (1) after causing a thrombophlebitis of the lateral sinus (an extension of which reaches the jugular bulb) passes mesially through the inferior petrosal; or (2) the thrombotic process primarily originates in the superior petrosal. These latter are far more common than is generally recognized, for the petrosal sinuses receive blood from small branches of

the tympanum which pass directly through the bone.[8]

In both petrosal types the invasion of the cavernous sinus is apt to be slow, and consequently the classical symptoms of exophthalmos, chemosis, etc., if present at all, are of transient duration; the establishment of the collateral venous circulation preventing obstruction to the return venous flow. Later, a phlebitis of the ophthalmic veins or of the sinus wall may cause chemosis and exophthalmos.[9]

CASE 14.

*Chronic cavernous sinus thrombophlebitis from the superior petrosal without involvement of the jugular bulb.*

*Synopsis: Absence of exophthalmos, chemosis, or swelling of lids until five weeks after onset, and then secondary to abscesses in both orbits, the result of retrograde thrombophlebitis of the ophthalmic veins; prior to which the only localizing symptoms of cavernous sinus phlebitis were irregular temperature, pain in teeth and dilatation of retinal veins, with negative blood cultures and a low leucocyte count* (7000).

Patient of Dr. George J. Holmes.

*History:* About one year previous the patient had discharge in the right ear for two weeks. No further ear symptoms until about three weeks ago, January 29, when the pain recurred accompanied by discharge, which has continued.

*February 18, Examination:* Ears: "Granulations in middle ear, right. Tenderness over right mastoid, slight deafness. Left ear negative. Nose: Polypoid degeneration and

[8] Rodger, T. Ritchie (in *Homeon's Article Jour. L and O.*, June, 1922, p. 282) reports cases of primary petrosal thrombosis.

[9] Ultman's case, in which no thrombosis was found in the sinus at post-mortem.

discharge from under both middle turbinates. Heart: marked mitral systolic murmur.

*Diagnosis:* Acute exacerbation of chronic right mastoiditis. Bilateral chronic ethmoiditis."

*February 20:* Operation (G. J. H.) "Simple mastoid operation. Perisinus abscess; no pulsation. Streptococcus and staphylococcus infection of mastoid."

During next week slightly irregular temperature: 98° F. in morning, but 100.1° to 101.2° F. at night. Pulse, 72 to 92. Sleeps well. No pain.

*February 27:* Temperature normal this morning, but 102° F. in the afternoon. Complains of pain in ear. Had quite a severe spontaneous hemorrhage from the mastoid wound, controlled by packing.

*March 4:* Temperature has continued normal in the morning, but 102° F. every night, and on one occasion reached 103° F. Perspires easily.

*March 4 to 9:* Around ward. Believed to be doing well. Temperature never over 100° or 101° F.

*March 11:* Pain in ear, which at times is severe. Pain in lumbar region. Slight cough; chest negative.

*March 14 to 17:* Throbbing pain in right side of head, and teeth; does not sleep well. Still irregular temperature, 99° to 101° F. Two teeth extracted.

*March 19:* Stiffness of neck.

*March 20:* Severe sore throat. Double optic neuritis, with outlines obscure; and retinal veins dilated; vision: right, 15/60; left, 15/30.

*March 22:* Pain behind eyes. Still complains bitterly of stiffness and pain in neck, and spine, with inability to sleep. Blood: leucocytes 7,120, polynuclears 73 per cent., lymphocytes 20 per cent. Second blood culture, negative; Wassermann, negative. Left hospital against advice.

During night pain in his head and neck became more severe; also slight pain behind his eyes, and in right arm and leg.

*March 23:* (51st day of disease) Right eye began to "bulge," and the next day the left also became prominent.

About the same time there appeared a swelling in the right side of neck. No vomiting, but choked when swallowing, at which time he felt as if he "had pus in his throat." Since leaving the hospital has had two chills and sweats; has had "bad spells," during which he was in a drowsy stupor and then became irrational; wanted to get out of bed, and "now does not use his right arm as well as formerly."

*March 26:* Complains of numbness in right arm and leg; "cannot feel with fingers of right hand."

*March 28:* Readmitted to St. Barnabas Hospital.

*Examination:* Somewhat irrational and in stupor. Edema of the neck on left side below jaw and along jugular.

*Eyes:* Double exophthalmos and slight chemosis; left greater than right; no nystagmus. Bilateral papilledema. Right retinal veins very full; cannot tell arteries from veins; no hemorrhages. Left veins full but not tortuous.

*Ears:* Right mastoid wound healed. Hearing: cannot determine, but mother states that he has been much deafer the last few days. Left ear, acute effusion.

*Upper Extremities:* Right, slight anesthesia of hand; right arc incoördinate. Left spontaneous past-pointing deviation.

*Legs:* Complains of stiffness of right foot. Apparently can move both legs equally well, but right incoördinately. B. P. 130-70. Temperature, 103.2° F. Pulse, 100.

Lumbar puncture: fluid cloudy greenish and under pressure; cell count 500; Fehling's not reduced; sterile.

*Diagnosis:* "Cavernous sinus thrombosis, following a lateral sinus thrombosis, extending through the inferior petrosal and down the jugular vein, with an intercurrent brain abscess and complicating localized meningitis."

*Comment:* Although it was evident that the man was suffering from meningitis; as there were no microörganisms in the fluid from the lumbar region, it was decided to explore the cavernous sinus, in the hope that the meningitis was of the protective type and would subside if the infected venous trunk could be drained.

*March 29, Operation:* (Patient in very poor condition. Took ether badly, rapid respiration and cyanosis through the whole operation). Exploration through the usual Gasserian ganglion route; terrific bleeding from small veins; middle meningeal artery obliterated by plugging of the foramen spinosum. Second branch of fifth nerve-root exposed; opening made through dura just below, and internal to the trifacial; followed by escape of cerebrospinal fluid. No pus, but a large amount of venous blood.

*March 30 to April 1:* Complains of pain in head, back of neck, and left ear, sore throat and difficulty in swallowing. Expectorates mucus, at times bloody. Sleeplessness; delirium; profuse perspiration. Exodus.

*Post-mortem:* (Dr. F. Sutton.) *Skull:* Right mastoid contains pus. Abscess in apex of both orbits. The cavernous sinus had not been opened as the operation tract had not entered the sinus, but had perforated one of the large vessels running from it.

*Basal Sinuses:* Right cavernous sinus thrombosed. Left thrombosed; circular thrombosed. Right superior petrosal, bloody pus. Inferior petrosal of the right side thrombosed, no pus. Superior and inferior petrosals of left side contains small clot at a little distance from the cavernous. Both lateral sinuses free, without evidence of thrombosis.

*Comment: Path of Infection:* From the post-mortem findings the infection had apparently travelled as follows: From the right mastoid, it entered the superior and inferior petrosals, traversed the cavernous sinus of both sides, and occluded by a clot the inferior and superior petrosals of the opposite side, where they enter the cavernous.

*Meninges:* Anterior surface of the pons covered with a thick layer of pus, also some pus on lower surface of the cerebellum.

*Brain:* Abscess of the left cortical motor area contained about 1½ ounces of bloody pus, apparently communicating with the cortex near the longitudinal sinus.

*Comment:* From the history and the autopsy, it is evident that the infection continued for sixty-two days, from

the beginning otitis, and forty days after the original mastoid operation (at which time the patient was probably suffering from a thrombophlebitis as shown by the operative findings, the irregular temperature and subsequent spontaneous hemorrhage).

It is impossible to fix the exact date of the extension of the cavernous sinus from the superior petrosal, but localizing symptoms of perivascular infiltration of superior petrosal and cavernous sinus thrombophlebitis (pain in teeth) occurred 6 days before death; followed three days subsequently by a basal meningitis, as shown by the stiff neck and papilledema; while the symptoms warranting a diagnosis of cerebral abscess followed two days later, as shown by the semi-numbness and paresis.

There was no exophthalmos until the formation of the abscess in the apex of both orbits from an extension forward into the ophthalmic veins from the cavernous sinus, which occurred at least eleven days after the cavernous sinus had become infected.

(c) **Extradural Abscess from Deep Cell Involvement.**—Examination of the literature demonstrates than an extradural abscess in the recess between the middle and posterior fossæ, from caries of the deep cells, is frequently associated with thrombophlebitis of the superior petrosal which, extending mesially, ultimately involves cavernous sinus thrombosis. (Cases 15 and F).[10]

From this the inference may be drawn that a deep extradural abscess having been found at operation, continuation of the septic symptoms should cause the surgeon to investigate the petrosal or cavernous sinus and not to confine his exploration to the lateral sinus and jugular bulb.

[10] Maybaum's case—Davis case, Wallace, 1895. (Poulson's case abstr: by Ballance.)

CASE 15.

*Acute septic sinus thrombophlebitis of aural origin following occlusion of petrosal sinuses, from caries of the perilabyrinthine cells with deep extradural abscess. (No ocular or neighborhood symptoms.) Autopsy.*

Patient of Dr. S. H. Baldwin.

*Previous History:* Left chronic otitis media with acute exacerbations.

*Present Illness:* Three weeks previously the ear stopped discharging. Past two days, severe pain in ear with dizziness and mastoid tenderness.

*December 2, 1919:* (Admitted to Eye and Ear Infirmary.) During night had a severe chill. Temperature, 107° F., and then gradually descended until next morning it was 102.4° F. Blood: leucocytes 18,000; negative. Lumbar puncture; cell count 86, globulin very slightly increased; sugar reduction normal; no organisms.

*December 3:* Another chill. Temperature, 104.3° F.

*Operation:* (Dr. S. H. Baldwin.) Mastoid opened. Sclerosed bone and granulation tissue. Thrombophlebitis of descending portion of lateral sinus.

During the next few days irregular temperature from normal to 104° F.

*December 6:* Complained of pain in the wound; chill; following day temperature reached 105° F. Blood culture (second) negative.

*December 8:* Two chills. Temperature continuously high, 103.2° to 106° F. Ophthalmoscopic examination, negative, except slight dilatation of veins of left fundus.

*Operation:* (W. P. E.) Grunert's operation, exposing the jugular bulb. No bleeding from inferior petrosal sinus. Could not demonstrate opening of inferior petrosal into bulb.

*Post-operative:* Transfusion. Blood: leucocytes 56,000, polynuclears 93 per cent., lymphocytes 7 per cent., culture, negative.

During next twenty-four hours the patient had two

chilly sensations, temperature continuously high, 102.4° to 106° F.; pulse, 82 to 118. Complained of pain in head.

Blood culture (4th), negative. Second lumbar puncture: fluid under slight pressure; cell count, 28; globulin slightly increased; Fehling's reduced; no organisms.

*December 11:* Severe chill. Complained of pain below right ear. Blood culture (fifth), negative.

*December 12:* Slightly irrational. Vomited. Severe chill. Temperature, 101.8° to 105.2° F. Leucocytes 25,000, polynuclears 85 per cent., large lymphocytes 15 per cent.

*December 13:* Delirious.

*December 14:* Death, fourteen days after beginning of acute exacerbation.

*Post-mortem:* (Dr. F. A. Sutton.) Body markedly jaundiced. Small extradural abscess on posterior surface of left petrous; with local area of bony caries near the posterior semi-circular canal, the dura here being slightly separated from this bone. Jugular bulb contains small amount of greenish pus. Septic clot in left lateral sinus, which extends backward to about 1¾ inches from torcular. Left superior and inferior petrosals thrombosed. Right cavernous sinus contains small clot not obliterating the sinus. Basilar plexus and the anterior spinal veins passing through the foramen magnum are thrombosed.

Viscera negative, excepting for acute parenchymatous changes from sepsis.

*Tissue Examination:* Section of infected sinus shows organized central thrombosis; vasa vasorum thrombosed. Nerves in cavernous sinus show edema and some leucocyte infitration, although during life there was no oculo-motor paralysis or facial pain.

*Comment:* The extradural abscess and bony caries of the perilabyrinthine cells probably contributed to the formation of the inferior and superior petrosal thrombosis by obliteration, which may explain why the inferior petrosal was not demonstrable at operation.

## CAUSE OF THE SYMPTOMS IN CHRONIC (COMPENSATORY) AURAL CASES, IMPORTANCE OF INFLAMMATORY INFILTRATION OF SINUS WALL.

When the infection enters the sinus from behind and causes its gradual obliteration, exophthalmos rarely occurs; however, in these cases, if the infective process extends forward and involves the orbital veins, there will be a late development of exophthalmos. A careful study of the neighborhood symptoms should make a diagnosis possible without chemosis, edema of the lids or exophthalmos; as a 3rd, 4th or 6th nerve paralysis, in the presence of an otitis with irregular temperature and chilly sensation, high leucocyte count (especially if accompanied by a papilledema without a high cell count in the lumbar fluid), can allow of no other interpretation. (Cases 2 and 14.) One positive blood culture should confirm the diagnosis.

In the chronic compensatory cases oculo-motor nerve paralyses are not due to pressure but to inflammatory infiltration and edema into the nerve fibres,[11] as they pass in the external wall of the sinus, between the reflection of the carotid sheath and the dura. Anatomically, the 3rd, 4th and 6th nerves are thus actually within the outer sinus wall; consequently, even a slight inflammatory infiltration is apt to cause an ocular paralysis. If, to these neighborhood symptoms with chronic pyemia, there is added *an abscess* of inflammatory infiltration of an area supplied by one of the veins running into the sinus, a diagnosis sufficient for operative action is surely warranted.

[11] Blake: *Amer. Jour. of Ophthal.*, January, 1921, and Hutchinson's case.

One of the cases herein reported (Case 2) was regarded as a purely ophthalmic case, and although for several weeks before the fatal termination the patient had the typical Gradenigo syndrome, running ear and paralysis of the 6th of the same side, the aural origin was entirely overlooked. It was only on the appearance of a retro-pharyngeal swelling that a diagnosis was made.

## ORBITAL ABSCESS AS A COMPLICATION OF CAVERNOUS SINUS THROMBOPHLEBITIS OF AURAL ORIGIN.

Many of the ear cases terminate with orbital abscess from the thrombophlebitis passing from the cavernous sinus forward into the orbit. (Cases 11, 14 and 20.)[12]

This latter pathological condition may be of clinical importance, for if an aural case with late ocular edema, swelling of the lids and exophthalmos, is to be attacked surgically, not only should the sinus be opened, but the abscess at the apex of the orbit should be drained.

### BIBLIOGRAPHY OF AURAL FROM PETROSAL SINUS CASES

Beyer, Dr. H.—"Ueber isolierte thrombus des sinus petrosus inferior."
Passow and Schaefer, Beiträge, etc. Bd. IV, H. 6. Monatschr. F. Ohrenheilk., 1912, 59.
Death from meningitis following acute mastoiditis and pyemia with metastases.
*Autopsy:* Disclosed inferior petrosal sinus thrombosed. Other blood vessels intact.

Blake, Eugene M.—Cavernous sinus thrombosis complicating meningitis. Microscopic studies. Amer. Jour. Ophth., 1921.

[12] Blake, Eugene: "Cavernous Sinus Thrombosis Complicated with Meningitis," Microscopic Studies, *Amer. Jour. Ophth.*, Series 3, No. 4, p. 16, 1921. Case of aural origin had ophthalmic thrombosis and inflammatory infiltrate into both orbits; also Charlin's case.

Braun, Alfred.—Case of cavernous sinus thrombosis complicating suppurative labyrinthitis.
Ann. Otol. Rhinol. and Laryngol., June, 1914.
Child seven years. Discharge from ear for five years. Died after two days' acute illness. Autopsy showed pus and granulations in right auditory meatus. Necrotic tract to petrosal sinus, which contained septic thrombus extending into right cavernous and across to left cavernous sinus. Perforation of the pia, covered with exudate.

Charlin, Charles.—Clinical observations of thrombophlebitis of the cavernous sinus and of the ophthalmic veins.
Ann. D'Ocul., Nov., 1920.
(Abstract in Internat. Med. and Surg. Survey, Dept. Ophthal. and Laryngol., Feb., 1921, 8a.)

Davis, H. J.—Specimen of temporal bone from a case of thrombosis of the cavernous sinus. Proc. Roy. Soc. Med., London, 1/3, Otol. Sec., p. 27, 1910-11.
Primary thrombosis of the superior petrosal from extradural abscess to opposite ophthalmic.

Engelhardt, G.—Pathology and treatment of cavernous sinus thrombosis following facial furunculosis and otogenic infection. Reports bilateral orbital abscess secondary to cavernous sinus thrombophlebitis of otitic origin from the inferior petrosal. Archiv. für Ohren- Nasen- und Kehlkopfheilkunde, Vol. 112, p. 272, 1925.

Lewis, Robt., Jr.—A case of mastoiditis complicated by thrombosis of left lateral, petrosal and cavernous sinuses. Operation, autopsy.
Arch. of Otology, Vol. 36, p. 910, 1907.
"A large parietal clot (white) was found in the right lateral sinus near the knee. A purulent thrombus was present in the left inferior petrosal and cavernous sinuses, more advanced in the latter. All the small veins collateral to the left lateral sinus were thrombosed—evidently the result of obliteration of the sinus. The body of the sphenoid was discolored, and the left sphenoidal cell was filled with a mucopurulent material."

Passow's Case.—(Ballance, Tr. 9th Inter. Otol. Congress, p. 314.)

After mastoid operation had tonsillitis; lateral sinus, normal at operation. Inferior petrosal septic. Passow thinks inferior petrosal was originally non-infective, but became infected from tonsillitis.

Poulson Case.—(Ballance, Tr. 9th Inter. Otol. Cong., p. 316.)

Otitis media, R., death in 16 days. No ocular symptoms. Autopsy. Extradural abscess. Thrombus superior and inferior petrosal. No thrombus of lateral or superior longitudinal. Little pus near internal meatus. Apparently entered from the posterior surface of petrous pyramid into the inner part of the petrosal.

Powell.—(Discussion of Davis Case, Proc. Roy. Soc., London, 10/3, Otol. Sec., p. 27, 1910-1911.

Invasion superior petrosal from necrosis of petrous.

Rodger, T. Ritchie.—Jour. Laryn. and Otol., 1921, Vol. 36, p. 169. Also referred to by Hemmeon, Jour. Laryn. and Otol., June, 1922, Vol. 37, No. 6, p. 281.

*Case 1:* Blood contained pneumococci. Wormlike clots came from the region of the superior petrosal. No autopsy.

Wallace.—St. Thomas Hospital Reprints, 1895, XXIV, 353.

Duration, 27 days. Autopsy: Dilated thrombosed vein, running from the apex of the petrous bone to the right sphenoidal fissure. Extradural abscess.

# CHAPTER V

## SPHENOIDAL CASES

### SPHENOIDAL (MESIAL) ORIGIN.

**Frequency.**[1]—The immediate cause of the cavernous sinus thrombophlebitis of sphenoidal origin may be:

(1) From osteophlebitis of the sphenoidal veins. (Case 16.)

(2) From caries of the body of the sphenoid. (Case 20.)

(3) From subinfections in which there is a combination of chronic sphenoiditis, which is brought into activity by an intercurrent suppurative process in the ear or throat. (Case 2.)[2] (See Superimposed Infection.)

In reviewing my cases and those in the literature, I am struck with the frequency with which an acute intercurrent infection (such as tonsillitis or otitis media) has brought into activity a latent chronic sinusitis (such as sphenoiditis or ethmoiditis), the recrudescent infection apparently giving rise to thrombophlebitis of the cavernous sinus.

[1] Boenninghaus: *Hand. bl. Spec. Ohr. und Chirurg.*, pp. 237-239, 240. Out of thirty cases of cavernous sinus disease due to nasal sinusitis, twenty-four originated from sphenoid and six from the frontal sinus.

[2] Chisholm and Watkins: Cases 9 and 10.

Cases 2, 14, 20 and 24 all had both otitis and sphenoiditis and were of the chronic compensating type. Case F had a combination of acute tonsillitis, acute otitis and chronic sphenoidal disease.[3] [4]

The cases of cavernous sinus phlebitis of sphenoidal sinus origin may be divided into:

(1) Acute toxic (septicemia). (Case 16.)

(2) Chronic compensatory with acute exacerbations. Chronic pyemia. (Cases 2 and 20.)

Case 16.

*Cavernous sinus thrombophlebitis originating from sphenoidal and ethmoid suppuration by an osteophlebitis of the veins, manifested by general toxemia without any localizing symptoms of cavernous sinus involvement.*

Patient of Dr. Walter L. Liefeld.

*Previous History:* The patient had headache on top of his head for years; generally occurring at three o'clock in the afternoon. Profuse discharge from nose. Intranasal frontal and ethmoid operation was followed by relief of headaches, but discharge from nostrils continued.

*September 7:* Suddenly an intense pain in head with a chill and temperature 101° F. Few hours later some rigidity of neck, which disappeared during night.

*September 8:* Cough and continuously high temperature (104° F.) during last twenty-four hours, with perspiration. Severe pain in back and head. No rigidity of neck.

*Examination:* Left nostril, profuse purulent discharge from chronic sinusitis. O. E. slight indistinctness of the margin of both discs; lumbar puncture: fluid slightly increased; bacterially negative.

*September 10:* Cough; slightly irrational, complains

[3] O'Malley's case (referred to by Smith) had tonsillitis and sphenoidal caries.

[4] Jacques and Lucien's case had osteomyelitis of sella and apex of petrous precipitated by a peritonsillar abscess.

chiefly of pain in head, abdomen and back. Continuous high temperature, 104° to 106° F. Blood: leucocytes 22,000, polynuclears 91 per cent., small lymphocytes 8 per cent., eosinophiles 1 per cent.; culture, negative. Lumbar puncture: no increase of fluid, cells 28, lymphocytes 70 per cent., polynuclears 30 per cent., Fehling's reduced; globulin increased; culture, negative.

*September 11:* Complains of pain in left side of abdomen. Great restlessness. Second blood culture, negative.

*Impression:* "It seems to me we can rule out a meningeal involvement. I am inclined to think the man has a blood stream infection although two blood cultures have been negative. It is possible in view of his chronic sinusitis that he had a cavernous sinus thrombosis with a subdural meningitis. I can see no reason why he should not have a septic phlebitis of one of the sphenoidal veins, although he has no symptoms whatever of sinus thrombophlebitis."

*September 12:* Herpes, cough, expectoration slightly bloody. Third blood culture, negative.

*September 19:* For past week the case has been regarded as one of pneumonia because of cough, increased respirations, and expectoration. Today intense pain in abdomen. Delirious and semi-comatose. Exodus (12th day after invasion).

*Post-mortem:* (Drs. Gray and Sutton.)

Body had been embalmed.

*Head:* Both sphenoidal sinuses are filled with a disorganized firm membrane, the result of a suppurative process. The bone above the sphenoid sinuses on both sides is apparently not affected. Ethmoids infected, but not so marked as the sphenoids although ethmoidal cells of the right side contain free pus. The left frontal sinus contains a mass of thickened membrane. It is, however, in the sphenoids that the great mass of grumous material is present.

The cavernous sinuses on both sides are filled with a brownish mass, undoubtedly the result of purulent infection although hardened from the embalming fluid.

*Abdomen:* A large amount of purulent exudate; omentum adherent to the distended intestines because of fibropurulent exudate. Thick yellow pus present in pelvis and left abdominal cavity. Liver and spleen contain numerous abscesses and septic infarcts.

*Tissue Examination—Head:* (Dr. John W. Gray.) "Sections from the frontal and the sphenoidal sinuses composed chiefly of pus. That from the cavernous sinus contained many pus cells; and that from the longitudinal sinus contained some pus. Pus is less abundant in sections from the cavernous sinus than from the frontal and the sphenoidal sinuses, and less abundant in the longitudinal than in the cavernous. Bacteria only found in the secretion from the sphenoidal sinus. None of these are arranged in chains."

*Abdomen:* Bacterial examination, streptococcus in chains, from general suppurative peritonitis.

*Comment:* Although there may be some doubt because of the body being embalmed, the result of the post-mortem suggests that death was due to an acute sepsis from acute exacerbation of a chronic infection of sphenoid, ethmoid and frontal sinuses. The infection from the sphenoids extending by way of the veins, passed through the bone into the basilar sinuses, and was followed by metastatic abscesses in other parts of the body—liver, spleen and kidneys.

**Symptoms of Sphenoidal Sinus Suppurations.**—Localized pain in the top of head, a violent pain associated with sphenoidal suppuration which comes and goes, was present in three of the seven cases. In one (Case 16) the cavernous disease originated direct from the sphenoid, and two others while of aural origin had a complicating sphenoidal sinus. (Cases 2 and 20.) From these three cases I am inclined to regard a localized pain in the top of head in the mid-line in the presence of sepsis as diagnostic of sphenoidal sinus

disease, the same as a localized nocturnal pain in the temporal region just above the ear is indicative of dural irritation when an aural suppuration exists on the same side. In one case there was an abscess in the naso-pharynx (Case 2).

Sphenoidal sinus disease was only diagnosed before death in one case (Case 2), although in three there was a constant expectoration (Cases F, 2 and 16), and in two, the pain and difficulty in swallowing (Cases F and 2) should have directed attention to the sphenoid and to the pterygo-maxillary fossa.

## SYNOPSIS OF BIBLIOGRAPHY OF SPHENOIDAL CASES

Blake, E. M.—Amer. Jour. Ophth., '21. Cavernous sinus thrombosis complicating meningitis. Five days' duration. Drum membranes of the ears apparently normal. Swelling of the left eye, and proptosis.

*Post-mortem:* Thrombosis of left lateral sinus, centered in the posterior part of straight sinus, and the posterior part of the longitudinal sinus. Both cavernous sinuses filled with thrombi. Also left ophthalmic vein. Center disintegrated, and showed large number of polymorphonuclear cells. At periphery thrombus was intact, in fibrinous strands forming dense network. Numbers of mononuclear cells, and few polynuclears, surrounded the vessels. Some areas of necrosis. Orbital cultures, staphylococcus. Brain, streptococcus, and staphylococcus. (May have been a sphenoid case.)

Chisholm and Watkins.—"Twelve cases of thrombosis of the cavernous sinus."

Arch. Surg., Nov., 1920, 483-512.

Case 5: Acute cold. Recrudescence of chronic sphenoiditis. Swelling of left eye and left side of face. Streptococcus.

*Post-mortem:* Antrum, sphenoid and ethmoid suppuration which may have caused orbital abscess.

Coupland Case.—(Quoted by Ballance.) Tr. of Ophth. Soc., Vol. VII.

Severe supraorbital neuralgia and drooping of left upper lid. Lasted four months. Deafness in left ear. After two months right lid drooped and severe right supraorbital neuralgia. Diplopia. Later proptosis, ophthalmos, chemosis. Insensate cornea. Discs and retina normal. Ptosis of left upper lid. In January, two months later, supraorbital pain had disappeared. Some proptosis of left eye. None of right. Right external paralysis and pain in right side and over right lid.

*February 18:* R. eye no movement. Discs reddened and veins larger than usual. Continued at work until February 27th. Became unconscious. Impaired sensibility left side of face.

*March 12:* Death.

*Post-mortem:* Meningitis. Pituitary body enlarged. Left cavernous sinus occluded by non-purulent clot. Right cavernous, circular, and transverse with purulent clot. Dura mater over sphenoid abnormally loose. Petrosal and other sinuses empty. Tympanum and mastoid cells no inflammation or pus. Source of infection unknown. Left cavernous sinus had healed, but right later became infected.

Horne, H.—"Cavernous sinus thrombosis due to organisms of the streptothrix."

Ann. Otol. Rhinol. Laryngol., Dec., 1913. Mentioned by Davis in the Proc. Roy. Soc. of Med., 1912-1913. Referred to by Dorland Smith, Arch. Ophth., 1918, XLVII: 482.

Hoston, K.—Thrombosis of the cavernous sinus. Norsk. Mag. f. Lægevidensk., Kristiania; 1921, LXXII:559.

*Case I:* Pain over eyes. Purulent secretion from nose. Eyes protruded, paresis of facial muscles. Death.

*Section Diagnosis.* Inflammation of the sphenoidal sinus associated with osteomyelitis, necrosis and gangrene of basilar sphenoid and occipital regions. Thrombosis of the cavernous sinus and ethmoid. Meningitis purulenta.

Pulmonary abscess. Parenchymatous degeneration in viscera.

*Case II:* Started in neck. Pain on opening mouth. Death in five days.

*Section Diagnosis:* Angina phlegmonosa. Thrombosed cavernous sinus. Osteomyelitis and necrosis of sphenoid and occipital bones. Meningitis purulenta. Edema of the meninges.

Hubby.—"A case of internal pachymeningitis, pansinusitis, cavernous and lateral thrombosis." Laryngoscope, Vol. 31:1204, 1921.

Discharge from nose one year. Thrombosis only about 12 hours previous to death. Cerebrospinal fluid under pressure. Duration about 10 days. *Autopsy.*

Jessop, W. H.—"Two cases of proptosis associated with a disease of ethmoid and sphenoid respectively." Tr. Ophth. Soc. U. K., Vol. XXIII, 1903, p. 184.

Key-Aberg, Hans.—"Thombosis of the cavernous sinus." Acta Oto-Laryngol., 3:37, 1921, Stockholm.

*Post-mortem:* Revealed meningitis in the ventricles and in the subarachnoid cavities at the base, especially in the posterior cavum cranii; no exudate over the hemispheres. After removal of the infundibulum there was found an irregularly shaped lesion the size of a farthing, to the right of the anterior wall of the sella turcica. This perforation had blood-stained edges, and opened into the sphenoidal cavity, which still contained a not inconsiderable quantity of pus, mixed with blood and coagulation. Thrombosis was present in the cavernous sinus on both sides and in the ophthalmic vein on the right side.

Rodger, T. Ritchie.—Case 4, Jour. Laryngol., April, 1921, p. 171.

"From frontal sinus, ethmoids and antrum, also sphenoid." 20 days' duration. No chemosis of either eye, but swelling over left eye, redness of conjunctiva and paralysis of external rectus. 3 days later distinct swelling over right eyelid.

*Ibid:* Brit. M. J., 1922, II, 461.

*Case I:* From sphenoid. Enlarged glands of the neck and below the lower jaw on the right side. Three days later purulent discharge from the nose, and high temperature. Right antrum full of pus. Middle turbinal amputated. Recovery.

*Case II:* Case was very similar to Case I, except that the onset seemed to be due to an acute infective rhinitis. No autopsy allowed.

Ortmann.—"Observations on intradural abscess and cavernous sinus thrombosis." 1890.

Referred to in Dreyfus: Die Krankh. des Gehirns, p. 40. Gives microscopical examination of cavernous sinus thrombosis from sphenoidal sinus. Résumé given in Turner, p. 70, Third Int. Laryngol.-Rhinol. Cong., Berlin, 1911, p. 71.

The transmission was through the periostitic and osteitic channels of the sphenoidal sinus; there was no evidence of lymphatic infection. The infection went through the small venous channels of the bone into the sphenoidal sinus and opening into the cavernous blood spaces.

Thomson, St. Clair.—"Cerebral and ophthalmic complications in sphenoidal sinusitis."

Brit. Med. Jour., July-Dec., 1906.

Month's duration. Post-mortem. He has collected 16 cases of involvement from the sphenoid with autopsies.

*Ibid:* Ophth. Rev., Vol. 27, p. 296. Two cases from sphenoid.

Turner, A. Logan and Reynolds, F. Esmond.—Furuncle of the right nasal vestibule. Septic thrombosis of the cavernous blood sinuses. Leptomeningitis. Death. Autopsy. Jour. Laryngol. and Otol., Vol. XLI, No. 2, February, 1926, pp. 73-86.

Whitehead.—Jour. Laryngol., XIX, Ap., 1904, p. 176, referred to by St. Clair Thomson. A fatal case of necrosis of the sphenoidal and posterior ethmoidal cells, phlebitis of the cavernous sinus; pyemia.

Jour. Laryngol., Vol. XIX, April, 1904, p. 179.

Quoted by St. Clair Thomson, Brit. Med. Jour., 1906, ii, 768.

# CHAPTER VI

## TONSILLAR AND NECK CASES

### CAVERNOUS THROMBOPHLEBITIS FROM INFECTIONS OF THE NECK

**Tonsillar Group.**—Cavernous sinus phlebitis of tonsillar origin may reach the sinus:

(a) By means of a primary septic retrograde thrombophlebitis of the tonsillar veins extending into the jugular vein; from which it ascends through the inferior petrosal to the cavernous (Case 17), or

(b) By way of the pterygoid plexus.[1]

(a) *From Thrombosis of the Veins of the Neck.*—Surgically we should regard with suspicion any continued or remittent temperature after operation on the tonsils or neck,[2] as retrograde thrombophlebitis which ascends into the jugular bulb, is much more apt to occasion an infection of the inferior petrosal than when the infection descends from the lateral sinus into the jugular bulb. The slit-like opening of the inferior

[1] Davis case.

[2] Crowe, S. J.: "Direct Blood Stream Infection Through the Tonsils," *Arch. of Internal Medicine,* April, 1924, Vol. 33, p. 473, gives illustrations of the "Thrombosis of the blood vessels that occur in localized ulceration of the epithelium in the crypts of a diseased tonsil."

Tanaka, M., and Crowe, S. J.: "Direct Blood Stream Infection Through the Tonsils," *Archives of Otolaryngology,* May, 1925, Vol. I, pp. 510-520.

petrosal into the jugular bulb is generally obliterated by an aseptic protective clot in a descending process [3] (Case 17).

CASE C.

*Suppurative thrombophlebitis of lateral sinus, from infected glands of the neck by a retrograde thrombophlebitis of the internal jugular vein.*

Patient of Dr. M. A. Miele.

*History:* Glandular swelling in neck from sore throat, associated with slight pain in ear but no discharge. Incision of suppurating mass and removal of enlarged cervical glands. Operation followed by pyemic temperature, chills and sweating. Ear, negative; hearing, normal. No cranial symptoms, but during the second week the patient suddenly developed a marked papilledema on the side of the suppurating wound.

*Operation:* Lateral sinus exposed in skull and found filled with pus. No bleeding from below—resection of thrombosed jugular in neck, the lower end of which terminated in the fistulous tract left by the original glandular operation. Prompt recovery.

[3] Woodburn, J. J.: "Cavernous Sinus," *Med. Press*, January 4, 1922, p. 13.

Case II. General sepsis from cuts on arms. Symptoms of meningitis and exophthalmos 14 days before admission. Reddish area around cut on neck. Areas of erythema over neck, arms, and chest. (Probably septic rashes.)

*Post-mortem:* Fluid contained in the lateral sinus had pus cells. Cavernous sinuses were found to be filled with thick blood-stained pus. Pus in meninges. Cortex, site of generalization of acute congestion. Spleen considerably enlarged. Staphylococcus pyogenes aureus in cultures from brain, cavernous sinuses, spleen, and heart's blood.

"The sinus infection may have spread from the rash around the neck wound through the angular and ophthalmic veins. The meningitis and septicemia were secondary. Or it may have been that an infection of one of the wounds about the arms or neck resulted in a septicemia and meningitis, causing thrombosis of the cavernous sinus."

## DIFFICULTIES OF DIAGNOSIS IN SEPTIC THROMBOPHLEBITIS OF THE JUGULAR VEIN IN NECK INFECTIONS.

Tenderness in the neck is usually not present in thrombophlebitis of the jugular vein, although in periphlebitis of the internal jugular there is frequently a perivascular infiltration present along the line of the vein itself. When, however, the infection originates in the tonsil the deep situation of the jugular in the pterygomaxillary fossa prevents the infiltration being felt; and in descending abscess formation within the jugular vein itself that follows the breaking down of the protective clot, there is generally no demonstrable jugular infiltration. (Case F.)[4]

In Case F the side diseased was not even suspected, although the free pus extended from the cranium into the chest; or in Case 17, in which pus retrograded by the vein from the neck into the lateral sinus, or in another case,[5] in which it was also an accidental operative finding.[6]

The Crowe-Beck [7] sign—filling of the retinal veins on compression of the carotid and jugular of the unaffected side—has been of assistance to me in several instances in diagnosing an obliteration of the suspected vein. The continuous pressure on the intact jugular by sand bags and head turning, which was the

[4] Hastings, Hill: Thrombosis of the Superior Thyroid Vein from a Cervical Abscess, between Larynx and Thyroid. Diagnosis made at Autopsy. *Laryngoscope,* Vol. 35, No. 6, pp. 423-429, June, 1925.

[5] Eagleton, W. P.: "Vestibular Tests in Intracranial Surgery." Case Burns. *Tr. Am. Laryn. Rhinol. and Otol. Soc.,* 1922.

[6] Gulline: *Jour. Laryn. and Otol.,* 1921, p. 127, reports a latent lateral sinus thrombophlebitis with involvement of the jugular vein, the only symptom being pain in left tonsil on swallowing.

[7] Crowe, S. J.: *Bulletin of Johns Hopkins Hospital,* Vol. 23, pp. 321-323, 1912.

cause of an ocular edema (Case 17), should have ensured a diagnosis of cavernous sinus obliteration.

CASE 17.

*Pyemia from ascending septic thrombophlebitis, originating in septic sore throat and adenitis involving the internal jugular vein and retrograding into the jugular bulb, lateral sinus, petrosals and cavernous sinus.*

(*The only symptom of venous obstruction during the course of the disease being a chemosis of the ocular conjunctiva on turning the head in the opposite direction during operation; a symptom of cavernous sinus thrombosis not previously described.*)

*Previous History:* Severe infection of hand several years previously; tonsils had been enucleated four years prior to fatal illness.

*History:* Acute swelling of glands of right side of neck associated with slight redness of mucous membrane of throat. For three days did not feel sick, so continued at work. Swelling of gland under jaw rapidly spread; culture from throat, streptococcus.

*First Operation:* Deep incision just below angle of jaw, evacuated a small amount of thin pus; wound packed with gauze. Few hours later, difficulty in swallowing.

Operation followed immediately by high temperature and marked edema of the submucous tissue of the tonsillar pillars of the right side extending into soft palate (such as is seen in peritonsillar abscess); no edema of the larynx. The edema subsided rapidly after removal of the pack from the external wound and entirely disappeared in two days.

Continued pyemic temperature, 99° to 105° F., with perspirations but no chills. Blood culture, streptococcus hemolyticus.

*On fifth day* after neck incision, severe chill. Complained of a pain in the calf of the right leg.

*Second Operation:* Jugular ligated low down in neck; free bleeding from lower end of jugular. Upper end not

investigated as there was *no tenderness* or infiltration at the upper part of the neck where previous incision had been made; or in pterygomaxillary fossa, which was carefully examined many times, both externally and by mouth.

Both neck wounds healed promptly but pyemic temperature continued with positive blood cultures. First transfusion.

*Third Operation:* Abscess of leg incised, during operation accidental injury to a large vessel in intramembranous space produced severe hemorrhage, necessitaing firm packing. Second transfusion.

A few days later a secondary hemorrhage from wound in leg necessitated amputation, after a third transfusion had restored his vascular system. (Examination of the amputated limb showed a long area of thrombophlebitis in the intramembranous vein.)

During *fourth week* continued septic temperature without pain. Repeated transfusion in an attempt to control blood stream infection.

*Fifth Week Impression:* "In view of the fact that he had an edema of his throat the day after the primary operation on his neck, which could only be accounted for by an involvement of the superior constrictor muscle, and as no other focus of infection can be found as the cause of the streptococcic blood stream infection, it is probable that he has a primary thrombosis of his jugular vein in the stylomaxillary fossa which has extended upward into the jugular bulb and lateral sinus; although there has never been any localizing symptoms, infiltration or tenderness surrounding the vein, and the original neck wound is entirely healed. The eyes, throat and ears are negative; sensorium was perfectly clear until yesterday; since when he has been slightly delirious during sleep but rational on awaking; pyemic temperature, chills and repeated positive blood cultures being the only evidences of illness."

*Fifth Operation:* (W. P. E.) Incision of lateral sinus evacuated free pus with foul odor from the lumen; aseptic clot adherent to the sinus wall extended upward to one

inch beyond the knee, where free bleeding occurred. *No bleeding from below,* although exposure extended well downward toward bulb. Within an hour of the beginning of the operation both ocular conjunctivæ became markedly edematous. As the patient's head was turned to the side away from the wound, the neck being extended by a sand bag thus pressing on the carotid and the jugular of the opposite side, this was believed to be the cause of the bilateral chemosis; which disappeared within a few hours after straightening his head.

Death during the sixth week, in coma from meningitis.

*Comment:* As there was no bleeding from below, the inferior, as well as the superior, petrosals must have been thrombosed; the infection having extended into the cavernous sinus causing its occlusion at the time of the last operation, as the pressure on the veins of the opposite side by the sand bags and the turning of the head obstructed the remaining venous pathways.

## BIBLIOGRAPHY OF TONSILLAR CASES

Chisholm and Watkins.—"Twelve cases of thrombosis of the cavernous sinus."
Arch. Surg., Nov., 1920, 483-512.
Case 12. Followed tonsillectomy.
*Post-mortem:* Cavernous sinus thrombosed, and basilar meningitis. Jugular vein free.
Note. Infection must have entered through pterygoid plexus.

Davis, H. J.—"Cavernous sinus thrombosis following tonsillitis."
Proc. Roy. Soc. Med., 1912-1913.
Tonsillar. 18 days' duration. Neck incision negative. No post-mortem, but D. thinks invasion was through facial vein.

De Lapersonne.—Phlébite suppurée des veines ophth. et des sinus caverneux. Arch. d'Ophth., Paris, 1885, Vol. V, p. 436.

Goodman.—"Primary thrombosis of jugular vein from tonsillar infection."
Ann. Otol. Rhinol. & Laryngol., June, 1917, p. 527.

Jacques and Lucien.—Soc. Franc. d'Oto-Laryngol., Mai, 1908.
(Referred to by St. Clair Thompson, Ophth. Rev.)
Cavernous sinus thrombosis from peritonsillar abscess. Death in 3 days.
*Post-mortem:* Osteomyelitis of sella and apex of petrous.

Jessop, W. H.—Trans. Ophth. Soc. of the U. K., Vol. XXIII, 1903.
Throat, infection of. 35 days' duration. Post-mortem.

Kernan, John D.—"Cavernous sinus thrombosis, secondary to peritonsillar abscess."
Laryngoscope, Vol. 31, 1921.
17 days' duration.
*Post-mortem:* Tonsillar abscess extended to the outer side of superior constrictor in the pharyngo-maxillary fossa. Internal jugular vein thrombosed. Thrombosis extended into lateral sinus, short distance beyond the knee. Both petrosals and both cavernous sinuses contained clots which extended into ophthalmic veins. All clots partially broken down. Abscess of left temporal lobe. Mastoids normal.

Mosher, Harris.—Tr. Am. Laryngol., Rhinol., & Otol. Soc., 1920, 327.
Throat; four weeks; patient died under operation; no autopsy.

O'Malley Case.—In Davis discussion, Proc. Roy. Soc. Med., 1912-13. Otol. Sec.
Abstracted by Dorland Smith, Arch. Ophth., 1918.
Followed adenoid and tonsil operation.

Passow.—Monatschr. für Ohrenheilk., 1912, p. 59.
Six weeks after healing from acute mastoid operation.
*Autopsy:* Septic thrombus as large as a pea found in the inferior petrosal sinus.
Referred to by Ballance; Tr. 9th Otol. Cong.

Tollens, C.—Angina and phlegmonous pharyngitis followed by purulent thrombosis of the cavernous sinus and purulent basilar meningitis. Arch. of Otology, 1904, Vol. XXXIII, No. 6, p. 487.

Vieu, G.—Sur l'opportunité de la ligature de la jugulaire dans la thrombophlébite du sinus latèral et de la jugulaire. Thése, Montpellier, 1912, No. 8.

## EXTENSION ALONG THE VEINS OF THE PLEXUS OF THE INTERNAL CAROTID ARTERY.

The venous plexus of the internal carotid originates in and is continuous with the cavernous sinus above; it surrounds the internal carotid artery in the carotid canal as the plexus descends, and empties into the internal jugular vein below, after receiving branches from the middle ear.[8]

A retrograde thrombophlebitis from either the middle ear or the jugular bulb may thus reach the anterior portion of the cavernous sinus by the carotid venous plexus without involvement of the petrosals.

**Cases of Infection of the Venous Plexus of the Internal Carotid Artery in the Carotid Canal of the Skull.**—There are five cases reported (Goldthwait,[9] Hett,[10] and Brunner[11] in addition to the two herein

[8] Spalteholz, Werner: *Handatlas der Anatomie des Menschen,* 1913, p. 445.

[9] Goldthwait, R. H.: *Mil. Surg.*, 1921, Vol. XLIX, p. 430.

[10] Hett, G. S.: *Pro. Roy. Soc. Med.,* 1910-11, *Sec. Otol.*, p. 28.

[11] Brunner, Hans: In three cases has demonstrated microscopically an infection of the veins of the carotid plexus in cavernous sinus thrombophlebitis of otitic origin without involvement of the petrosal sinuses. He believes that this mode of infection frequently occurs, as out of twenty-two autopsies on cases of lateral sinus phlebitis at Alexander's clinic in Vienna during the last two years, twelve were found to have had a cavernous sinus thrombophlebitis; either by way of a phlebitis of small veins secondary to the menin-

reported—Cases D and 24) in which the infection apparently extended along the plexus of veins around the internal carotid artery.

Brunner's microscopical studies have, however, demonstrated that his mode of infection is very frequent although rarely recognized. Both acute and chronic cases of this type may possess certain clinical features, as:

The early and transient appearance of an exophthalmos in a pyogenic type of venous sinus infection of aural origin—especially if associated with a pharyngomaxillary abscess of the lateral and posterior wall of the nasopharynx—may enable a diagnosis of cavernous sinus infection by way of the venous plexus of the carotid canal.

The carotid plexus enters the cavernous sinus in its forward portion, at a point mesial to the entrance of the emissary veins which pass through the foramen ovale and which perforate the foramen lacerum [12] near the entrance of the ophthalmic vein. The foramen lacerum is situated anterior and mesial to the carotid foramen and above, posterior and external to the Eustachian tube.[13] It transmits a vein which, when present, opens below into the scaphoid fossa of the skull.

The coalition of the carotid plexus, the accessory

gitis, or from a carotid plexus thrombophlebitis. Beiträge zur Kenntnis der otogenen Cavernous Phlebitis. Fifth Congress of German Otological Society, Munich, May 28-30, 1925. *Centralblatt für Hals, Nasen, und Ohrenheilkunde,* 1925, Band VII, Heft 12, p. 684.

[12] *Zuckerkandl, Atlas d. topogr. Anatomie d. Menschen,* Kopf und Hals (Fig. 45), and *Corrosionspräparat des Sinus cavernous* (Fig. 46), pp. 49-50.

[13] *Quain's Elements of Anatomy,* pp. 86-87.

veins of the ovale and the small medial situation of the vein of Vesalius with the ophthalmic veins, in reality forms the arms of the anterior "lake" of the cavernous sinus; Zuckerkandl [14] depicts the carotid venous plexus and the emissary of the ovale as coalescing before entering the cavernous sinus. Phlebitis of the carotid venous plexus would consequently produce early exophthalmos, but because of its small size and the numerous collateral circulations, the proptosis is apt to be early and only transient, recurring later with complete obliteration of the cavernous and ophthalmic.

Case D.

*Chronic venous infection of the carotid plexus followed by cerebral softening from arterial occlusion (precipitated by operative trauma).*

*Previous History:* Left chronic otitis since childhood; aural polypus having been removed several times. During last two years has had many periods of pain and several attacks of dizziness.

*Present Illness:* Four weeks previously suffered from "facial neuralgia," for which four teeth were extracted, without relief. One week prior to admission developed left-sided facial paralysis. Two days later had a chill, which was repeated on the following day.

*August 14:* Admitted to Newark Eye and Ear Infirmary, complaining of slight pain in back of neck and left facial paralysis.

*Examination:* Taste sense reduced on affected side. Ears: left drum membrane destroyed; *granulations apparently springing from floor of middle ear.* Hearing: left, whisper close to ear. Right, watch ¼ inch; whisper 2 feet.

[14] Zuckerkandl: *Loc. cit.*

*Vestibular Tests:* Rotation nystagmus normal. Cold caloric (left), gives nystagmus.

*August 16, Operation:* Radical mastoid exenteration. In hospital until September 4, during which time had periods of slight rise in temperature, but never above 100° F. No pain in head. Thought to be doing well when discharged.

*October 6:* Patient's memory has been very poor since operation as "he will talk about something, and a few hours later seems to have absolutely no recollection of it."

*Examination:* Slight spontaneous nystagmus, most marked on looking toward right (away from the suppurating ear). Lateral movement of head causes dizziness. Complete facial paralysis of left side. Taste, not affected. *Hearing:* Left, total deafness; Weber referred to unaffected ear. Left caloric produces no nystagmus. Temperature, 98.4° F.

*October 8:* Slight chill followed by sweat and left-sided headache. Temperature, 99.1° to 101.1° F. Pulse, 65-88. Leucocytes 12,400, polynuclears 60 per cent., lymphocytes 36 per cent., large mononuclears 3 per cent., transitionals 1 per cent. During night had another chilly sensation; and headache, which had been in left temporal region, became violent and involved right side, occipital and frontal. Pain made worse by moving head.

*Examination:* Very little dizziness on movement. Romberg, slight; sensation of falling toward affected side, not altered by change of position of head. Right pupil slightly larger than left. Fundus: margin of disc indistinct with marked optic neuritis of left. Coördination good. Patella reflex exaggerated.

*Diagnosis:* "Undoubtedly has suppurative labyrinthitis, and possibly a meningitis or a local intradural abscess."

Decided to open labyrinth and to explore posterior fossa.

*Operation:* Exposing floor of middle ear disclosed granulation tissue extending along both anterior and posterior walls of Eustachian tube.

Anterior bony wall of external auditory canal removed

to obtain room, which was considerably aided by opening jaw; *granulations surround carotid artery.*

Horizontal semicircular canal obliterated. During its exposure, accidental opening in dura, through which the brain herniated. Superior semicircular canal also obliterated; above superior canal was a necrotic area of dura covered with granulations; in the center of which a fistula led into the middle fossa. On touching the dura with a probe there was a loss of a large amount of cerebrospinal fluid.

Cochlea opened, liberating free pus. Modiolus accidentally broken, associated with further profuse escape of cerebrospinal fluid from lateral arm of cisterna pontis.

*Remarks on Technic of Operation:* The cochlea should have been opened at once. The modiolus would not have been fractured accidentally had not the landmarks been lost by complete removal of the canal system.

*Post-operative Course:* On coming out of ether the patient had a spontaneous nystagmus in all directions. Could not sleep, and complained of severe pain in head. Pulse, 88; temperature, 102° F.

Fifteen hours later, delirious; temperature, 106° F. Exodus (twenty-four hours after operation), temperature having risen to 108.2° F.

*Post-mortem:* Dura of middle fossa is covered with exudate.

*Basal Sinuses:* All large sinuses distended with blood; no clot in sigmoid sinus. Brain considerably injected, convolutions obliterated, brain overfilling the cranial cavity. No evidence of meningitis. Subdural hemorrhage on posterior surface of petrous bone on left side from operative trauma.

On removing brain considerable turbid fluid around medulla and pons.

On opening brain, whole mass edematous. No internal hydrocephalus, but ventricles contained turbid fluid. *Partial clot in internal carotid artery of left side;* and the brain substance has an area of greenish edematous appear-

ance, which starts at the posterior part of the internal capsule on the left side, and runs out toward the cortex for a distance of 2 cm. Small hemorrhagic extravasations on left side of pons.

*Post-mortem Diagnosis:* Meningitis from chronic labyrinthitis. Beginning cerebral softening from vascular occlusion. Edema of the brain precipitated by operation.

*Comment:* In view of the history, operative and post-mortem findings (and the microscopical studies of Brunner in somewhat similar cases), there can be little doubt that the infection from the middle ear involved the venous plexus of the carotid artery as early as July (as shown by the dental neuralgia, by the position of the granulations in the carotid canal, and the changed mental state following the primary operation). Later the infection extended into the facial canal, although the labyrinth was still not infected. In September, the labyrinth was invaded, associated with the development of a serous meningitis. The trauma of the excessive chiseling and curettement of the Eustachian tube during the October operation gave rise to the thrombus in the carotid, which was already partially formed from the infection of the carotid venous plexus.

In connection with Carotid Venous Sinus Infection, see too, Brock: Microscopical Findings in a Case of Apparently Simple Chronic Ear Suppuration, with a Fatal Outcome, Beitr. z. Anat. Physiol. Pathol. u. Therap. d. Ohres., etc., 1925, Vol. 22, p. 25.

## CHAPTER VII

### NON-INFECTIVE TYPE OF CAVERNOUS SINUS THROMBOSIS

As is to be expected, the proportion of recoveries from non-infective cavernous thrombosis is much larger than in the suppurative types. Accidental injury to the cavernous sinus during operation on the Gasserian ganglion for the removal of basal tumors or for fractures of the base, while presenting alarming exophthalmos and chemosis, as a rule has not ended fatally.[1]

CASE E.

*Traumatic arterio-venous ancurism. Retrobulbar ligation of dilated ophthalmic vein. Recovery.*[2]

*September 17:* Admitted to City Hospital in semi-comatose condition from an auto accident.

*Diagnosis:* Fracture of skull with multiple head and face wounds. Few days later (when facial swelling had subsided), discovered to have a slowly increasing right-sided exophthalmos and chemosis, with marked venous engorgement of right eyelids and conjunctiva. Eyeball immovable. Auscultation revealed a loud high-pitched bruit.

Fundus: Superior retinal vein and all its branches, many times normal size; with sausage-shaped appearance. Infe-

[1] Cushing, Hartley, Knapp and Hutchinson's cases.

[2] Eagleton, W. P.: A case of pulsating exophthalmos with ligation of ophthalmic vein. Reported to the Section of Ophthalmology, N. Y. Academy of Medicine, May 17, 1920. *Archives of Ophthal.*, Vol. 49, p. 638 (1920).

rior retinal vein similar, but to less extent. Arterioles, normal in size. No hemorrhages.

Pressure applied to eyeball caused an obliteration of all the fundus vessels, the disc becoming white (demonstrating a uniformity of arterial and venous pressure; probably from a venous arterial communication). Vision: Right, counts fingers at 3 feet.

The exophthalmos gradually increased until the lids no longer covered the cornea, which showed signs of ulceration.

*October 2, Operation:* Kroenlein osteoplastic flap exposing the retrobulbar contents. Very large tortuous vein presented—thought to be ophthalmic—obliterated by Cushing's silver clips, applied as far back toward apex of orbit as possible.

*November 22:* The exophthalmos began to subside immediately after the operation, and the chemosis has been reduced by massage.

*March 12:* Eyeball back to normal position; complete paralysis of external rectus (possibly from the operation).

Patient says that at times she hears a bruit in her head; auscultation, negative—although when she closes her eye she can hear the bruit, but when the lid is open it cannot be heard. It is the opening and closing of the eye that undoubtedly causes pressure on the blood vessels and gives rise to a bruit, as it is only heard as the eye closes, not as the lid opens nor when it remains closed.

"*Fundus:* Disc white; superior retinal veins are about the color of normal artery; superior retinal artery small; and can see an obliteration of its temporal branch. Inferior retinal artery and vein of normal size but entirely too red in color. Pressure on eyeball entirely empties the superior and inferior retinal arteries and a great part of the inferior retinal vein; so there is still some venous arterial communication."

R. V. 20/70. Loss for form in lower quadrant of field.

# CHAPTER VIII

## DIAGNOSIS

### SYNOPSIS OF DIAGNOSIS

*Symptomatology:* There are three distinct types of infection of the cavernous sinus: (1) one with outstanding symptoms of venous engorgement of the cavernous sinus; (2) another without such symptoms, but with a profound toxemia (high temperature, continued or intermittent presence or absence of chills and perspiration); and (3) a third with symptoms of chronic pyemia with the slow development of neighborhood symptoms. In the chronic compensatory (posterior) aural cases, too positive signs of cavernous sinus involvement must not be expected.

The diagnosis must rest on:

1. Known site of infection,
2. Blood stream infection,
3. Early but slight symptoms of obstruction:
   (a) Temporary fullness of veins of retina,
   (b) Crowe signs,
   (c) Temporary ocular edema (Case 17) from continued pressure on neck.
4. Neighborhood symptoms from involvement of the nerves in the sinus, by inflammatory edema or direct pressure; causing a paralysis or paresis of the 3rd, 4th and 6th nerves, or paresthesia or

anesthesia of the first branch of the 5th nerve manifested by
(a) Transient or permanent diplopia,
(b) Herpes of face and lips; pain in teeth or supra- or infra-orbital regions, or behind the eye.

5. Neighborhood abscess in the adjacent soft parts secondary to a retrograde septic thrombophlebitis extending from the cavernous sinus into smaller venous radicles, which normally empty their blood into the cavernous sinus or its immediate accessories; thus we can have abscess of the:
(a) Orbit (Case 15).
(b) Occiput (Case 20).
(c) Neck (Case 14).
(d) Nasopharynx (Cases 2 and 24).
6. Symptoms of complicating diseases:
(a) Headache and
(b) Papilledema from serous meningitis of basal cisterna.

## PHYSIOLOGICAL AND ANATOMICAL PRINCIPLES UNDERLYING LOCALIZATION

A. **Of Classical Symptoms.**—As previously emphasized, the early symptomatology depending upon the portal of infection will be influenced by the *sudden or gradual* occurrence of the occlusion of the sinus.

When the infection causes venous stasis within the ophthalmic vein, exophthalmos and chemosis result. (See anterior symptomatology.) When infection enters the cavernous sinus behind the entrance of the

pterygoid plexus as from the sphenoidal sinus or from the posterior end of the cavernous sinus from the petrosals, there are apt to be no pathognomonic symptoms.

However, analysis of my cases makes me believe that if they are carefully followed and repeatedly examined day after day, it will be possible to diagnose a cavernous sinus thrombosis, with but few exceptions.

B. **In the Chronic Compensatory Cases.**—In the absence of symptoms of obstruction, the localizing symptomatology will depend upon (1) a known focus of infection; that is, the presence of a suppurative lesion in or near the cranium, which (2) may possibly produce a cavernous sinus disease, associated with signs of a slowly advancing lesion from this site; the most important of these latter being (3) slight and often transient evidence that the blood stream is gradually diverted into other channels, and (4) the presence of neighborhood symptoms.

All of these considerations (often trivial in themselves), may make possible an early diagnosis during the only period which offers a possibility of relief from surgery.

1. **Considerations About Sinus Phlebitis in General; Diagnostic Importance of Known Focus of Infection.**—As the result of a large experience with intracranial suppuration, I am convinced that in the presence of a known focus of infection in the ear or nose, no other possibility should be permitted to draw the surgeon's attention away from the aural or nasal suppuration as the probable cause of the cerebral

symptoms. For, while intercurrent infections, such as pneumonia, frequently develop, the slightest acute exacerbation of the chronic process (earache, cessation of aural discharge, pain or swelling) should definitely decide the known focus as the origin of the cerebral complication. This conclusion should be reached, of course, in the absence of positive evidence of an intercurrent infection—as manifested by rapid respiration or physical signs in the chest, etc. In fact, I feel assured that many of the so-called intercurrent discases, especially pneumonia, have their immediate origin in chronic aural or nasal suppuration.

Again, while infection is generally obvious in discharging ears; sphenoidal suppuration is more frequently overlooked than recognized.[1] (Cases F, 2, 16, 20 and 24.)

Case 18.

*Cavernous sinus thrombophlebitis from unsuspected sphenoiditis.*

Patient of Dr. Henry Orton.

Severe headaches off and on for some time—no nasal discharge (probably sphenoidal sinus suppuration). Pain in teeth for many days. Sudden and severe pain in eye and head, with double vision. (Compensation cavernous sinus involvement.) Seen by ophthalmologist who advised extraction of teeth. Five extracted immediately, followed by exophthalmos and chemosis; high temperature, delirium, coma (secondary meningitis), and death five days later.

2. **General Blood Stream Infection.**—In suspected cerebral lesion of nasal or otitic origin, it is of the

[1] Yerger, C. F.: "Intracranial Complications of Sphenoidal Sinus Disease," *Illinois Med. Jour.*, April, 1923.

utmost importance to determine immediately the presence of a blood stream infection, as, in the early stages, a differential diagnosis can usually be made between sinus thrombophlebitis and infection of the meninges, or of the cerebral tissue itself. Later, however, such a differentiation may be impossible because two or all three lesions may coexist.

Case 14 had brain abscess and meningitis; Cases F, 13 and 17 had meningitis.[2] [3]

## DIAGNOSTIC IMPORTANCE OF REPEATED BLOOD CULTURES

The routine procedure, in cases of suspected blood stream infection of whatever source, should be to have a blood culture made daily. One blood culture may be as useless diagnostically as the taking of the temperature on one occasion only.

In many of the cases here reported, several negative blood cultures were recorded. (Cases 2, 14, and 15.)

One positive blood culture is diagnostic.

Similarly the leukocyte count is significant and should be made daily—(Case F, 21,000—40,000—52,000), as it is not only an indication of whether the infection is being controlled but also of the degree of resistance of the individual patient.

**Importance of Positive Blood Cultures as Localizing Symptom of an Involvement of a Large Venous Sinus.**—It has been impressed on me that localized

[2] Davis, E. D. D. Post-mortem specimens from a case of cerebellar abscess complicated by Thrombosis of the Lateral and Cavernous Sinuses. Proc. Roy. Soc., London, 1919-20, 68.

[3] Kernan's case had temporosphenoidal abscess.

abscess or thrombosis of a small venous radicle of the head or neck does not occasion a positive streptococcic blood culture without involvement of a *large venous trunk* (Cases 17 and F); and while cases of streptococcic blood infection with positive cultures from thrombophlebitis of small venous radicles in other parts of the body are reported in infections of the head, I have come to regard a positive blood culture as diagnostic of phlebitis of a large venous sinus.

Staphylococcic blood stream invasion may be different, as it seems to have a tendency to reach the blood stream by way of the small venous radicles, and having once gained control, the phlebitis steadily retrogrades. But from my limited experience in this type of case where a positive blood culture has been present, a carefully conducted post-mortem has always demonstrated an involvement of a large venous trunk.

Case F.

*Infective thrombophlebitis of the jugular vein and bulb (unlocalized during life) possibly originating from an ascending phlebitis of the neck (tonsils), associated with a purulent sphenoiditis, a suppurating mastoid (deep perilabyrinthine cells) of the opposite side and a general serous meningitis. Death from general sepsis. Autopsy.*

*Previous History:* Subject to tonsillitis. Always has phlegm in throat. Last year after cold, became hard of hearing and never entirely recovered it.

*Present Illness:* Three months ago, following a sore throat had pain in right ear; with further reduction of hearing, and discharge. During next six weeks had repeated paracentesis of drum because of continuation of pain. Maxillary antrum also drained.

*May 5-29:* Simple right mastoidotomy by Dr. Howe. Twelve days in hospital, apparently doing well.

*June 1:* "Contracted cold," since when has had severe pain on top of head, almost continuously. Irregular temperature.

*June 2-5:* Pain in head very much worse; nauseated; severe chill followed by temperature 104° F.

*June 6:* Admitted to infirmary. Appears very ill—looks septic; has a continuously high temperature, perspires profusely, and is constantly raising phlegm. "Feels weak and sleepy most of the time." Complains of nausea; violent pain in head; pain on left side of throat when swallowing; pain in right arm (which he does not move well because of stiffness of shoulder).

*Examination:* Right suppurating mastoid wound, without granulations; totally deaf in ear. Left hearing poor, but has been so for past year. No dizziness. O. E. both discs, tendency to slight blurring at margins. No rigidity of neck. No Kernig. Blood: leucocytes 20,400, polynuclears 84 per cent., small lymphocytes 11 per cent., and large lymphocytes 5 per cent.; cultures, streptococcus nonhemolyticus. Lumbar puncture: fluid clear, pressure slightly increased; cell count 222; polynuclears 78 per cent., lymphocytes 22 per cent., sterile.

*Preoperative Diagnosis:* Right lateral sinus thrombophlebitis and extradural abscess causing a serous meningitis (latter based on high cell count without bacteria in the cerebrospinal fluid).

*Operation:* Extensive superficial necrosis of squama just above external auditory canal and zygoma; caries in the solid angle. Dura of middle fossa bluish, thickened and covered by exudate which extended over tegmen. Free bleeding from vein entering sinus. Sinus opened low down toward the bulb; free bleeding. Small incision of dura liberated large quantity of bloody cerebrospinal fluid.

*Operative Diagnosis:* Serous meningitis, evidently result of infection from bony necrosis (possibly tuberculosis); serous labyrinthitis.

*June 6-8:* Patient had bad night, yellowish hue to skin (septic look). Expectorates thick yellow mucus. Swelling over right shoulder. Slight spontaneous rotary nystagmus on looking toward left (away from lesion); streptococcus hemolyticus in culture, Temperature, 101° to 104° F.

*Progress Note:* "Although he is toxic, I am fairly well satisfied with his condition, as he would be worse if he had a general meningitis. The swelling of the shoulder may be a toxic synovitis."

*Dressing Note:* "On exposing the wound, with patient in sitting position, can see the whole descending sinus collapse when he inhales a long breath; showing that the sinus, jugular bulb, and right jugular vein are free from obliterating thrombus."

*June 9-10:* Looks very septic. Restless, voids involuntarily, but is otherwise mentally clear. When he swallows, he also coughs. Temperature, 101° to 103° F. Blood: leucocytes 46,600, polynuclears 90 per cent., small lymphocytes 4 per cent., large lymphocytes 6 per cent.; culture, streptococcus in long chains. Lumbar puncture: fluid yellow, not under pressure, globulin increased, cell count 25, polynuclears 40 per cent., lymphocytes 60 per cent.; sterile.

As I believed that he must have a clot in the lateral sinus or the jugular bulb, I again opened the sinus; profuse bleeding.

*June 11:* Sometimes irrational, but always optimistic; says he "feels fine" (euphoria). Skin yellow; has a Kernig. Temperature, 101° to 103° F. Pulse, 120-140. Respiration rapid, 36-40. Lungs, negative. Liver slightly enlarged. Leucocytes 51,000. (Picture of profound sepsis.) Lumbar puncture fluid yellow, not under great pressure, cell count not high. Aspiration of shoulder failed to reveal fluid.

*Progress Note:* "He has the euphoria seen in meningitis limited to the frontal lobe, but has not the appearance of a general suppurative meningitis."

*June 12:* Profoundly septic. Slightly delirious. Streptococci in blood, although not in chains as formerly. Wound sloughing. Dura gangrenous and discharges a

large amount of watery secretion which looks like cerebrospinal fluid.

*Progress Note:* "Evidently has brain suppuration (encephalitis) or sinus thrombosis, but not a frontal lobe abscess. I am inclined to think that he has a local process near the wound (an internal pachymeningitis), as the cell count is not high, but the fluid is yellow."

*Comment in View of Post-mortem Findings:* All this time no attention was paid to the opposite (left) sinus or jugular as there was no swelling or pain, although when the patient first entered the hospital he complained of pain on left side of neck in swallowing, had a constant expectoration of mucus, and later swallowing made him cough. Probably if we had pressed over the right side, we might have seen ocular changes which would have made us think that the left jugular was occluded.

*June 14-15:* Distended abdomen, temperature continuously high; pulse fast and weak; rapid respiration. Thought to be dying.

As no local focus of infection was demonstrable, although he had a profound blood stream infection, he was transfused; 250 cc. of whole blood being introduced. This was followed by a chill, temperature, 106° F., then a sweat, then an acute fall of temperature to normal; a second rise to 107° F., and death from cardiac exhaustion 6 hours after transfusion.

*Post-mortem:* (Dr. F. Sutton.) *Basal Sinuses:* Right lateral sinus (exposed by operation) enormous in size and pervious; contains large post-mortem clot not adherent to wall. Extending down from the jugular bulb is a chicken-fat clot, which can be lifted out (macroscopically a normal sinus and jugular bulb). Left lateral sinus very small in size; its descending portion contains an adherent septic clot filling the whole sinus down to the jugular bulb. Pressure applied to neck over jugular vein forces out the clot, and pus wells up from the jugular vein below.

*Bones of Skull:* Remains of right petrous bone (operated side) free from infection, except an *area of perilabyrinthine*

*cells* at the apex of the petrous pyramid (where it abuts on the occipital and sphenoid bones just behind the carotid canal), which is filled with pus. (One of the observers thought the inferior petrosal sinus of the right side was infected, but of this there is doubt as the base of the skull directly over the sphenoidal sinuses and the adjacent cancellous portion of the basilar process of the occipital bone were soft and carious, although they contained no pus.) Left mastoid free and clear.

*Nasal Sinuses:* Both sphenoids filled with sero-pus; on removing which a hemorrhagic condition of the mucous membrane lining of the sinuses is disclosed.

*Brain and Meninges:* Upper surface of brain edematous; pia-arachnoid distended with fluid which is yellow, and has many fine small flakes of exudation following the convolutions generally, especially around the median line. (Undoubtedly, a serous meningitis with exudation; the leucocytes eliminate the infection of the meninges through the Pachionian villi.) Over the posterior part of the occipital lobe this edematous condition is marked; but here there are few exudations, only serum (cultures made from serum negative).

Exposure of the subdural space in the anterior portion of the brain (just posterior to the cribriform plate), discloses two yellowish masses; which can be scraped away without injuring the pia-arachnoid (localized meningitis of the subdural space). The posterior fossa contains multiple small hemorrhages, which extend along the anterior surface of the cord in the neighborhood of the basilar process of the occipital bone.[4]

*Neck: Left internal jugular vein contains free pus* through whole extent into chest. Right jugular vein has no clot.

*Chest and Abdomen:* Bloody fluid in left chest. No pleural adhesions. Left lung three-quarters consolidated.

[4] Eagleton, W. P.: "Purulent Inflammation of Basal Sinuses and Meningeal Cisterna," *Proc. College of Physicians,* Philadelphia, February 20, 1923, p. 420, Case V.

Right chest contains bloody fluid. Right lung, fifty per cent. consolidated. Beginning pericarditis. Liver normal.

*Cultures:* Smear of left jugular bulb, streptococcus. Smear of left lateral sinus, streptococcus. Culture of left lateral sinus, streptococcus and pyocyaneus bacillus.

Culture of right lateral sinus showed streptococcus and pyocyaneus (general blood stream and terminal infections). Smear and culture of serous exudate of brain, negative. Right and left sphenoidal sinus cultures contained streptococcus and pyocyaneus.

*The Outstanding Features of the Brain Post-mortem* were: (1) the serous meningitis with exudate, the exudate being confined chiefly to the region of the Pacchionian bodies in the median line with serum in the basal cisterna; (2) exudate in the subdural space over the sphenoidal sinus area (this must have been old); (3) a purulent sinus involvement of the left lateral sinus and its descending portion, coming apparently from below upwards.

*Discussion:* The most interesting problem is how the invasion got into the left descending portion of his lateral sinus and jugular bulb. It seems inconceivable that it travelled from the perilabyrinthine cell of the right side; for, although the inferior petrosal of the right side may have been affected, the cavernous sinus certainly was not.

The right lateral sinus and jugular bulb were enormous in size, while the left was abnormally small, and it is possible that this anatomical difference may have been a factor. For if the left inferior petrosal was the site of a thrombophlebitis (as one observer thought), and a septic infarct had descended from it into the jugular bulb, septic occlusion might have followed because of the bulb's small size and the ebb and flow of the blood within it. However, this does not seem probable. The sphenoidal sinus as the sole cause of left jugular bulb phlebitis is to be considered, as there were a subdural meningitis and caries of bone in the neighborhood; but without involvement of the cavernous sinus I do not see how it could occur. It is much more probable that the infection originated from a combination

of a chronic sphenoiditis and an infection of the left tonsil; for the patient had complained of catarrh for years, and for several days after his admission he apparently had some difficulty in swallowing, with pain on the left side of the throat; also when he took two or three drinks of water it would make him cough. All this pointed to an infection deep in the stylomaxillary fossa.

**Appearance of Patients in Blood Stream Infection.**—In blood stream infection, the patients, although recording a high temperature, with or without rapid excursions, look well—too well—in proportion to the temperature. A boy with lateral sinus thrombosis with temperature of 107° F. said to the writer, "If you will leave me alone I will get well."

**Resistance to Surgical Shock in Blood Stream Infections.**—A peculiarity in pyogenic blood stream infection is that the patients stand ether anesthesia remarkably well, at least until the time of myocardial degeneration from bacterial invasion of the heart muscle and valves. Although during operation large quantities of blood are frequently lost, in only one instance in the writer's experience has the patient died as a result of the operation. The patient had a septic myocardial lesion. (Case 10.)

There is apparently something associated with a blood stream infection that causes certain changes within the blood, which gives the individual not only a sense of well-being but of greater resistance as far as surgical manipulation is concerned. They are, in fact, much better surgical risks than the average normal individual. This may appear to be a very broad statement, but it is undoubtedly true. The

tolerance of diseased individuals to certain specific drugs—mercury, quinine, etc., has long been recognized.

When, however, the blood-producing tissues themselves are attacked (such as in leukemia), any operative trauma may precipitate a fatal termination in infective processes. I have had uniformly disastrous experiences in several cases of pyogenic infections in leukemia; these apparently demonstrate that the diseased leucocytes are devoid of their normal reparative and protective qualities; the inflamed and infective tissue undergo necrosis from nutritional death without suppuration, consequently any operation is immediately followed by a rapid extension of the infection. That the terminal infection travels largely by way of The venous system is suggested by the following case.

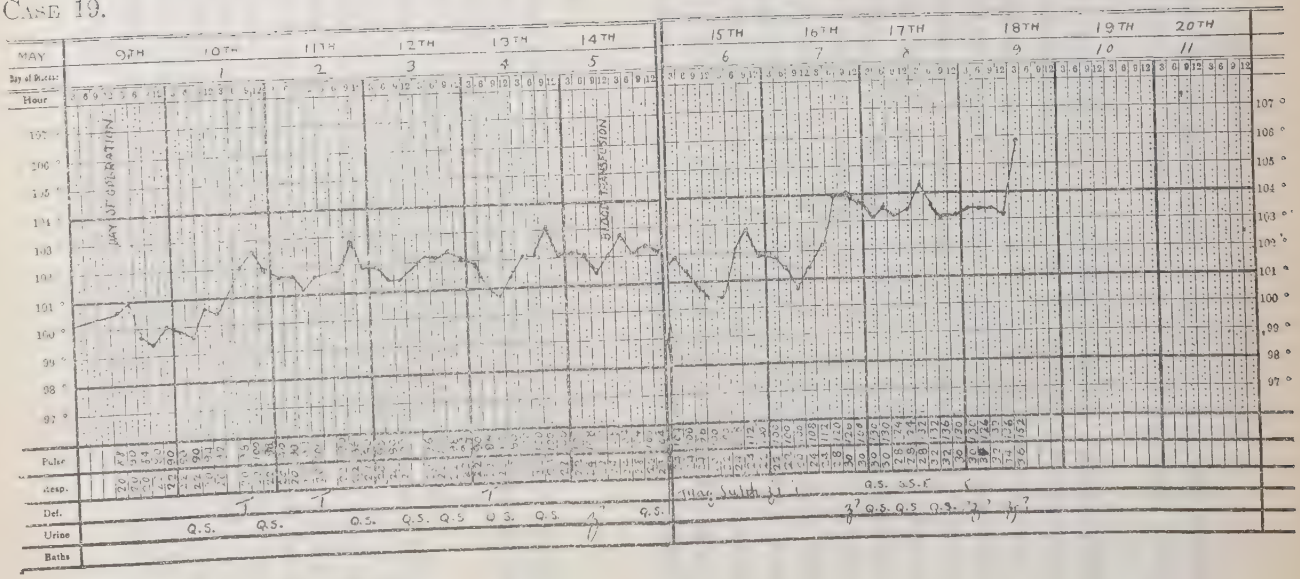
Case 19.

*Cavernous sinus thrombophlebitis precipitated by operative trauma from chronic antrum and oral infection in a patient suffering from myelogenous leukemia.*

Seen in consultation with Dr. W. O. G. Quinby.

*Previous History:* Six months previously had several teeth extracted from upper jaw; followed by what was diagnosed as "general sepsis from his teeth." Had very little pain or temperature; but has been unable to work for past three months, gradually growing whiter, with "gastric disturbance." Six weeks previous to admission had five more teeth removed from left upper jaw (novocaine used). A swelling now developed about the middle of the hard palate, which quickly ulcerated with a foul odor to the discharge, but no pain. The process gradually spread to

CASE 19.


MAY
9TH
10TH
11TH
12TH
13TH
14TH
15TH
16TH
17TH
18TH
19TH
20TH
Day of Disease
Hour
DAY OF OPERATION
DAY OF TRANSFUSION
Pulse
Resp.
Def.
Urine
Baths
Q.S.

the left side of the palate, the right side healing within a month.

*Examination: May 3:* Patient cachectic and weak; perspires freely. Edema of left side of face, particularly over upper jaw; some puffiness about upper eyelid.

*Mouth:* Ulcer the size of a five cent piece on left side of the hard palate with exposed necrotic bone; through which a probe may be passed into antrum.

*Nose:* Pus in left nasal fossa.

*Eyes:* No exophthalmos. Cornea and media clear; fundi normal but very pale. Vision, both 20/30; fields contracted.

*Diagnosis:* Chronic purulent sinusitis of left antrum.

*May 9:* Admitted to Eye and Ear Infirmary. Somewhat delirious.

*Operation:* (W. O. G. Q.) Incision of anterior wall and curetting of antrum; no pus liberated, but whole area is necrotic. Smear from antrum shows bloody pus, cocci and bacilli. Culture from pus: streptococcus. First blood culture, negative.

*May 10:* Patient very weak and drowsy; restless; muttering and twitching almost continually while sleeping. Temperature ranges from normal to 102° F. Blood: hemoglobin 35 per cent.; reds 2,300,000; whites 83,000; neutrophile myelocytes 70 per cent.; normoblasts and microblasts. Second blood culture, negative. Laboratory diagnosis: "myelogenous leukemia."

*Treatment:* Blood transfusion 500 cc. and two radium exposures over spleen. No reaction.

*May 14:* General bogginess of uvula. Left eyelid swollen, with exophthalmos and slight chemosis of outer portion of conjunctiva. Left fundus now has hemorrhage in different parts of retina, chiefly near disc. Right fundus: veins full, also small hemorrhages. Spleen enlarged. Temperature has been 102° F. since operation. Uranalysis: sp. gr. 1020; albumin, epithelial and granular casts.

*May 15:* Unconsciousness, alternating with restlessness. Patient complains of dizziness, and of pain in lower jaw

and left side of face. Perspires profusely. Third blood culture, negative.

*May 17:* Is irrational at times. Complains of pain in head. Opposite right eyelid swollen and ball proptosed. Temperature, 104° F.

*May 18:* Exodus in deep coma.

*Comment:* The cavernous sinus thrombophlebitis with its terminal meningitis was undoubtedly precipitated by the surgical intervention; the myelogenous leukemia precluding the normal protective reactions.

**Temperature in Blood Stream Infections.**—Preconceived ideas about the necessity for chills, sudden elevation of temperature followed by perspiration and remission (symptoms familiar in connection with lateral sinus thrombosis), must be abandoned in view of our present knowledge about pyemia, septicemia and sapremia.

Cases in which there is remission of temperature and chills, are undoubtedly less virulent than those in which there is a continuously high temperature.

My experience leads me to believe that, in a general way, a continuously high temperature with chills is the rule with free pus in large venous trunk (Cases F, 17 and J. B., see Eagleton, *loc. cit.*, case 4); while continuous temperature and headache indicate a meningeal complication in cavernous sinus thrombophlebitis. (Cases 15, 16 and 20.)

This may be of diagnostic importance because a continuously high temperature following jugular bulb thrombosis, with headache, points to a septic involvement of the cavernous sinus, possibly because of the meningitis (protective or suppurative) that always

accompanies a phlebitis of the large sinuses of the base. The compensatory cases all exhibit the usual remission of temperature to or near the normal with sudden rises. (Cases 2, 13 and 14.) Infants never, and children seldom, have a distinct rigor; but in children the sudden rise in temperature is associated with a slight blueness of the lips and fingers, and in infants is frequently preceded by a convulsion.

**Delirium.**—The appearance of delirium is of the gravest significance; as long as the infection is confined to the sinus the sensorium is entirely clear. Delirium doubtless does occur in cases of lateral sinus thrombosis that recover, but its presence is indicative, I feel sure, of a meningeal involvement—the usual terminal process.

The associated meningitis need not be suppurative and diffuse but may be protective and limited in type, in which case if the cause of the meningeal irritation can be removed, the inflammation within the dura is apt to subside. This favorable outcome cannot, however, be expected in cavernous sinus thrombophlebitis because of its inaccessible position.

Toxic delirium, which is so common in general infections—such as pneumonia, or following surgical operations for gall-bladder suppuration—does not occur in infective thrombophlebitis.

**Headache** is not present when infection is confined to the sinus itself. It signifies a meningeal irritation from perisinuous infiltration or suppuration of the bone in the vicinity of the meninges.

Cases 2, 16 and F, all had headache on top of the head with sphenoidal sinus disease and an associated serous meningitis. The pain in the frontal region (Case F) was undoubtedly due to the localized meningitis in the subdural space over the sphenoidal sinus.[5] Another case had an intense bursting headache with papilledema, showing meningeal involvement.

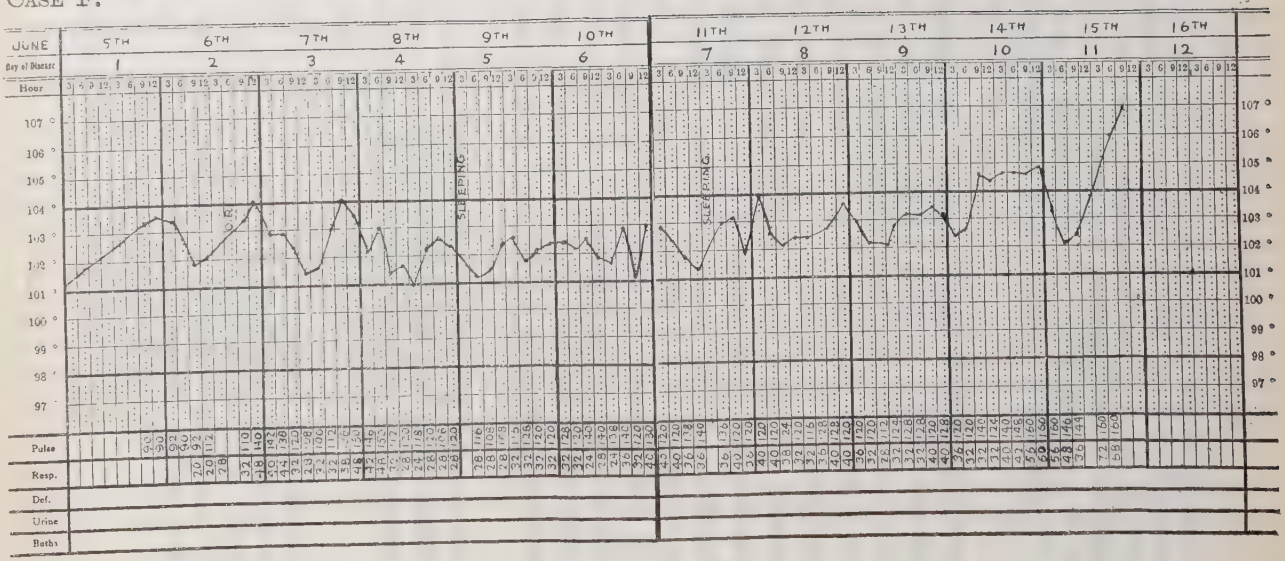
3. **Evidence That Venous Blood Current Is Being Obliterated.**—Because of the absence of valves in the ophthalmic and facial veins (see page 138), not only may infection ascend through them into the cavernous sinus, but they may be the route of a secondary descending thrombophlebitis; the infective process having originally reached the sinus from the petrosals (posterior cases), then descending into the ophthalmics, thus gives rise to proptosis and chemosis or an orbital abscess late in the disease. (Cases 3 and 17.) In such a case frequent ophthalmoscopic examination may disclose dilatation of the veins while the blood is still attempting to enter the cavernous sinus (Case 15).

There may be slight ocular edema due to the occlusion of the cavernous sinus before the other anastomotic veins have assumed the function, or a transient diplopia from edema or pressure.

In one case the patient had a transient chemosis from pressure on the neck by sand bags and rotation during operation (Case 17). In another, long before the appearance of edema of the ocular conjunctiva there was a dilatation of the conjunctival vessels (Case 7), such as is frequently seen in pulsating exophthalmos.

[5] See Coupland's Case.

## Case F.


JUNE
5TH
6TH
7TH
8TH
9TH
10TH
11TH
12TH
13TH
14TH
15TH
16TH
Day of Disease
Hour
SLEEPING
Pulse
Resp.
Def.
Urine
Baths

**Intraocular Signs.**—Papilledema occurs when the infection has invaded the basal cisterna.[6] [7]

Cases 2, 12, 14 and 22 had bilateral papilledema.

Temporary fullness of the veins of the retina may be an ocular manifestation. (Cases 15 and 23.) It may be transient because of the free anastomosis of the ophthalmics and their communications with the pterygoid plexus. As the ophthalmic and facial veins have few valves, they easily become the channels whereby the blood which usually enters the cavernous sinus is conveyed away from the orbit.

In one case,[8] operative obliteration of the lateral sinus and ligation of the jugular below the facial were immediately followed by intense optic neuritis, dilated and tortuous veins and numerous retinal hemorrhages, accompanied by total blindness and cord-like varices over the whole of scalp and chest, signifying venous thrombosis obliteration of the superior longitudinal sinus. The post-mortem revealed that the operation had obliterated the chief pathway from the head as the opposite jugular bulb was of very small size.[9] [10] [11]

While extensive occlusion of the venous pathways undoubtedly causes papilledema from venous

[6] Fundus Changes Occur in 20 per cent. of the Cases." Parsons, *Pathology of the Eye*, Vol. IV.

[7] O'Malley's case. Post-mortem showed osteitis of the sphenoid extending along optic nerve of one side.

[8] Eagleton: "Brain Abscess," Case 23, pp. 129-130.

[9] Passow's Case abstr.: by Ballance.

[10] Chisholm and Watkins: Case 8, recovery from ethmoidal disease. "Cord-like veins in scalp," showing establishment of collateral circulation, probably longitudinal sinus disease.

[11] Vernieuwe, *loc. cit.*, p. 59, reports sudden appearance of venous stasis of the papilla from thrombophlebitis because of an abnormal emptying of the longitudinal into the transverse sinus at the level of the mastoid emissary vein.

stasis, the uniform presence of headache in these cases makes me believe that the choked disc is partially of meningeal and cerebrospinal fluid system origin.

### REASON FOR ABSENCE OF INTRAOCULAR HEMORRHAGES IN CAVERNOUS SINUS THROMBOPHLEBITIS IN CASES OF INFECTION FROM OPHTHALMIC VEINS.

A septic thrombosis can exist in the ophthalmic veins at the apex of the orbit without retinal hemorrhage or without intraocular signs. This is due to the fact that the blood leaves the eyeball both anteriorly and inferiorly, there being free anastomosis of the veins anteriorly into the facial system, and inferiorly into the pterygoid plexus. When hemorrhage within the eyeball occurs, it signifies either that the central vein itself is involved (Case 11), or that pressure from the inflammatory process is so near the eyeball that the retinal circulation can no longer be taken care of by the anastomotic communications.

4. **Neighborhood Symptoms.**—Localized pain in top of head (see sphenoid cases). Pain behind the eye is suggestive of petrosal sinus thrombosis from irritation of the first branch of the trifacial.[12] [13] (Case 23.) In such cases anesthesia of the area supplied by the first branch might enable a diagnosis.[14]

In one case there was herpes of the lips, a sign of irritation of the Gasserian ganglion, which has an intimate

[12] Coupland's case.

[13] Robinson and Hutchinson's case.

[14] Key-Aberg: Acta Oto-Lary 3:37, 1921; patient had a numbness of homolateral side of face at beginning.

relationship to the cavernous sinus. (Case 16.)[15] [16] This may occur long before anesthesia of the cornea, a frequent accompaniment of the cases with extreme exophthalmos. One case had difficulty in swallowing (Case F), with return of fluid through the nose. Two had pain in the teeth (Cases 14 and 18).[17]

Ocular paralysis is generally the first sign of invasion of the cavernous sinus in the chronic aural cases. The paralysis is caused by the inflammatory exudate involving the nerves themselves as they pass in the cavity of the sinus.[18] (See microscopic report in Case 15.)

The long course of the sixth nerve makes it especially vulnerable to pressure, and in the posterior cases it is the first nerve to be paralyzed. It may be "pinched" as it passes over the apex of this petrous pyramid and under the inelastic petrosphenoidal ligament; or the inflammatory exudate of the sinus may compress it between the carotid and the bone. (See page 37.)

One case (No. 2) had paralysis of external rectus of homolateral eye and later of bilateral sixth nerve, and partial third of the opposite side.

When the purulent process is limited to the interior of the sinus—abscess within the vein—without an associated periphlebitis, neighborhood symptoms do not develop.

[15] See Leegaard, p. 152.

[16] Lombard's case (Ballance) had herpes.

[17] Chisholm and Watkins, and Uchermann report trismus from irritation of third branch of fifth nerve, also one case of unilateral sweating.

[18] See Illustration in Hutchinson case, *Trans. Ophth. Soc.*, U. K., 1888.

**5. Diagnostic Importance of an Associated Adjacent Abscess of the Soft Parts From Occlusion of a Venous Radicle Emptying Into the Cavernous Sinus.** —In the presence of a positive blood culture and an abscess of the soft parts situated in an area drained by one of the large venous sinuses of the head (such as the occiput or orbit), the surgeon should always consider the possibility of the abscess being due to a direct extension from an infected clot; thus the position of the abscess may enable the surgeon to locate the particular venous trunk affected.

Such secondary abscesses are too often dismissed as of metastatic origin; or still worse, regarded as the primary source of the blood stream infection. (Cases 2 and 20.)[19]

Five of my cases had deep abscesses of the soft parts adjacent to the cavernous sinus, all due to extension outwards of the thrombophlebitis of a large venous trunk into one of the small veins emptying into it, with rupture. Two (Cases 5 and 14) had pus in the apex of both orbits at post-mortem; one (Case 14) during life had a swelling in the neck. Two cases had an abscess in the nasopharynx; one (Case 24) overlooked, and one (Case 2) regarded as of sphenoidal origin. One (Case 20) had a deep abscess in the deep tissues of the occipital region.

Case 20.

*Acute infective cavernous sinus thrombosis of aural origin associated with sphenoidal sinus suppuration, early secondary abscess, but no ocular chemosis or exophthalmos until third week of disease, two days prior to death from general sepsis. Post-mortem.*

*Previous History:* The patient had several attacks of periodical insanity, the result of alcohol. Sunstroke last

[19] Jessop's case.

summer, since then headaches, generally in the occipital region, especially in warm weather, or when becoming excited. No previous aural disease.

*Present Illness, February 1:* "Caught cold with cough"; no discharge from nose, severe pain in head, referred to vertex or, according to wife, to left side of head. Next day had stiff neck and pain in both ears, but no discharge. During the last two weeks had temperature from 100° to 101° F., associated with two "nervous chills" and two attacks of vomiting.

*February 15:* Admitted to infirmary, complaining of headache and dizziness. Immediately after admission, slight chill, temperature, 104° F.

*Examination:* Tenderness and bogginess over both mastoids and entire back of neck, especially at the insertion of the trapezius and along the upper portion of the sternocleidomastoid muscle of the right side. (Two blisters have been applied to the back of the neck; stiffness thought to be due to this.) Tenderness over right mastoid antrum, right ear drum red, and seems bulging; hearing, whisper eight inches. O. E. negative, no nystagmus. Patellar reflexes exaggerated. No Kernig. No Romberg. Urine: Sp. g. 1030, contains large amount of albumin. Lumbar puncture: fluid clear; not under tension; sterile. Blood culture, negative. Paracentesis of right drum membrane; smear from aural secretion, diplococcus (probably pneumococcus).

During the next three days continuous high temperature, 103° to 105.2° F.; pulse, 90-120. Does not sleep well. Complains of headache.

*February 18:* Cough, delirious and irrational, with intervals of semi-consciousness; pain in back of head. Bogginess of right side of neck increased. Second blood culture, negative.

*Diagnosis:* "Beginning pneumonia."

*February 19:* Slight edema of right ocular conjunctiva; pain in eyes, severe pain in head and back.

*Preoperative Comment:* Because of the location of the

pain in the muscles of the back of the neck, where blisters had been applied, I had felt that the sepsis was not from the ear. However, as the patient had now become delirious and the bogginess in neck had increased, I decided to open the mastoid, although with several others I thought he was suffering from pneumonia, with acute nephritis; this because of the cough, continuously high temperature, and albuminous urine.

*Operation:* Abscess was located deep in the cellular tissue of the suboccipital region posterior to the right mastoid; the abscess extended backward to the median line; this abscess apparently originated from a suppurative osteomyelitis of the occipital bone. The right mastoid contained pus.

*February 20:* Right ocular chemosis; patient very delirious. Spontaneous profuse discharge from left ear. Temperature suddenly descended to 99° F., followed by rise to 105° F. Third blood culture, streptococcus.

*February 21:* Exodus.

*Post-mortem:* (Dr. Harrison S. Martland.) *Head:* Right eye presents considerable conjunctival edema. Scalp in the occipital region edematous; no hemorrhagic extravasations except a slight one over the right mastoid incision. Left middle ear and mastoid, pus and granulation.

*Sphenoids:* Both contain thick creamy pus, the right more than the left.

*Ethmoids:* Free and clear.

*Neck:* A superficial abscess is located below the mastoid, and a deep abscess over the lateral process of the upper four cervical vertebræ.

*Basal Sinuses:* A purulent clot extends posteriorly from the right transverse sinus nearly to the torcular, and inferiorly from the sigmoid sinus into the jugular bulb, through which it involves the jugular vein in the neck. The purulent thrombosis involves the right superior and inferior petrosal sinuses, the right cavernous and the anterior and posterior circular sinuses. The left cavernous sinus is also filled with a red adherent clot broken down into free pus in

places. From the left cavernous sinus the infection invades the left superior petrosal, occluding its inner portion by a pussy clot. The left inferior petrosal is free. The posterior vertebral basilar sinuses are all thrombosed. The left lateral sinus while markedly distended is free from thrombosis, as is the left jugular bulb. The superior longitudinal sinus contains separate foci of pus. The right ophthalmic vein is normal.

*Brain:* Convolutions flattened. Over the right hemisphere and along the longitudinal sinus can be seen numerous flakes of fresh fibrin. There is a slight amount of clear fluid at the base, but no signs of suppurative meningitis.

*Heart:* Acute parenchymatous degeneration.

*Lungs:* Left, septic bronchopneumonia with abscess formation of lower lobe. Right, septic bronchopneumonia with abscess formation of the lower lobe. Healed tuberculosis.

*Spleen:* Acute splenitis.

*Kidneys:* Left and right, nephritis (cloudy swelling and active hyperemia).

*Liver:* Slight productive hepatitis (fatty infiltration and degeneration).

*Primary Cause of Death: Streptococcic (short chained) sepsis, due to sinus thrombophlebitis of basal sinuses from localized suppurative sphenoidal sinus and mastoid disease.* Beginning suppurative meningitis, particularly over the cortex.

*Comment:* It is probable that the acute otitis was the cause of the extensive basal sinus purulent thrombophlebitis. But it is also possible that the associated chronic sphenoidal sinus disease contributed to the cavernous sinus involvement. The severe pain in the top of the head points to the latter condition.

# CHAPTER IX

## DIFFERENTIAL DIAGNOSIS BETWEEN SINUS THROMBOSIS, MENINGITIS, AND BRAIN ABSCESS

(a) **Sinus Phlebitis and Meningitis.**—With continuously high temperature and headache, especially if there are present irregular attacks of delirium, meningitis is to be suspected. It may be partially excluded by lumbar puncture, as it is now known that meningitis is apt to be confined to the basal cisterna in some cases for a considerable period (Case 13) before becoming general; during which time the cerebrospinal fluid from the lumbar region may, or may not, give evidence of the invasion of the leptomeninges, depending upon the extent to which the protective reaction has been able to localize the meningeal infection.[1]

There is, however, some danger of lighting up a meningitis by the performance of lumbar puncture during the active stage of a blood stream infection—that is, when the micro-organisms are free in the blood stream.[2] Consequently, if a lumbar puncture is to be performed, the smallest quantity of fluid should be

[1] Eagleton: "Meningitis; Its Surgical Treatment." Third communication. *J. A. M. A.*, December, 1924, *Tr. Sec. of Otolaryngology*, 1924.

[2] Eagleton: "Meningitis," first paper, *Tr. Amer. Otological Society*, 1911.

withdrawn that is consistent with a proper examination.

(b) **Brain Abscess.**—How shall it be excluded? Largely by absence of what has been designated by Dr. Harrison S. Martland as a protective meningitis; in which there is an increased amount of cerebrospinal fluid and a high cell count, chiefly polymorphonuclear.[3] (One case, number 14, had a complicating brain abscess.)

[3] Eagleton: "Brain Abscess," p. 160.

# CHAPTER X

## PROGNOSIS

It is evident that thrombophlebitis with a purulent clot in the cavernous sinus cannot result in recovery from surgery alone in a large proportion of cases, because of its anatomical inaccessibility and pathological relationship. The great depth from the surface and its mesh-like structure prevent complete drainage, such as is obtainable in thrombophlebitis of the lateral and transverse sinuses. It will always be impossible to eliminate surgically an infection which has extended from one cavernous sinus through intercommunication to the opposite side. In such an event, the slight hope of recovery must rest on surgery aided by the transfusion of immunized blood.

However, it seems probable that as long as the infection is limited to one sinus a certain proportion of cases should recover, provided the surgical attack is instituted at an early date, and the meningitis (which generally accompanies a periphlebitis) is either protective in type, or confined to the basal cisterna or the subdural space contiguous to the sinus.[1]

[1] Dwight and Germain in 131 autopsies found the thrombosis limited to one side in 31 cases. They made no distinction between a septic and aseptic clot. *Boston Medical and Surgical Journal,* Vol. 146, 1902.

Dwight and Germain,[2] in 1902, collected all the reported cases of cavernous sinus thrombosis and found that seven per cent. of them recovered spontaneously. Up to the present time, there is only one reported case of cavernous sinus thrombosis which has been cured by operation directly on the sinus itself (Bircher [3]), with the exception of Case 22 herein recorded, which was associated with a ligation of the common carotid. Case 23, of drainage of the primary phlebitic process in the lateral sinus and jugular bulb and the control of the deeper thrombosis of the cavernous sinus through rest by ligation of the common carotid, is also unique. All the other recoveries have either occurred spontaneously (Reden, Landsberg, Kolb), or from vaccines (Chisholm and Watkins, Case 12), or the operation has simply drained the original focus of infection. (Bourgeois, Rodera, Uchermann, Tewert, Johnson, Preysing, Boonacker, Brieger, Benninghaus reported by Ritter.)[4]

## BIBLIOGRAPHY OF REPORTED RECOVERIES IN AURAL CASES

Bircher.—Centralblatt für Chirurgie, 1893, p. 483.
Case four months' duration; recovery. Chronic inferior petrosal infection. No exophthalmos, but ocular paralysis. Recovered because abscess at the apex of the petrous pyramid was evacuated. Clot in cavernous sinus probably aseptic.

Boonacker.—Ritter's Case, Zeitung f. Hals-Ohr-Nasenheilk., 1, 1922, 352.
From ear. Chronic panophthalmia. Clas. symptoms. Recovery.

[2] Dwight and Germain: "Thrombosis of the Cavernous Sinus with Report of Four Cases Including One Cranial Operation," *loc. cit.* "The literature includes 178 cases aside from those herein reported, and of these 182 cases only 14 recovered."

[3] Bircher: *Centralblatt für Chirurgie,* November 22, 1893.

[4] Smith, Dorland, *Arch. Ophth.,* 1913, Vol. XVIII, p. 482, says that there are twenty-three cases of recovery, but they overlap.

Bourgeois, H.—"Otitic thrombophlebitis of the cavernous sinus. Cure."
Ann. des mal. de l'oreille et du Larynx., 1908, 34/2:397.
Patient had obstruction to venous return from a deep cervical abscess with probably an aseptic clot in the cavernous sinus.

Brieger.—Early operation on a phlebitis (without clot) of the lateral sinus probably stopped the process. Arch. Otol., 1896, p. 365.

Colledge.—Brit. Med. Jour., ii, 1922, p. 460.
(Four recoveries.)

Day, E.—"Supposed Cavernous Sinus Thrombosis."
Ann. Otol. Rhinol. Laryngol., Vol. XI, 1902, p. 520.
Chronic infection from ear. Recovery with loss of sight in both eyes. Secondary abscess in neck.

Dighton, Adair.—"Cavernous sinus thrombosis of otitic origin." Recovery.
Ann. of Otology, Rhinol. and Laryngol., Vol. XXI, p. 411.
Examined fifty cases in literature since 1883 with no recoveries. His case was acute case of venous blocking, following secondary operation for closure of posterior skin wound after radical mastoid. Three days later thrombosis of lateral and jugular and sigmoid. Immediate relief of basal symptoms, but continued metastatic. Recovery. The acute infection was relieved through the jugular vein. The clot in the jugular vein and lateral sinus was originally quiescent, but was lighted up by the operation.
*Ibid.* The Practitioner, 1913, Vol. X, p. 562.

Hemmeon, J. A. M.—A case of cavernous sinus thrombosis of otitic origin, by supposed infection of the petrosal sinuses.
Jour. of Laryngol. and Otol., Vol. XXXVII, June, 1922.

Johnson, H. R.—"Cavernous sinus thrombosis of otitic origin, with recovery."
Laryngol., Vol. XXII, 1912, p. 124.
Acute mastoid operation. One week later general infec-

tion. Four days later operation. Jugular resected, and lateral sinuses opened. Atrophy of optic nerve.

Kolb.—Berlin. Klin. Wchnschr., 1876, p. 663.
Note: Certainly an aseptic thrombus, with detachment of emboli into retinal vessels, as the patient had no rise of temperature during the entire three months of illness.

Preysing.—Arch. Otol., N. Y., 1898, XXVII, 340-354.
Note: Symptoms of venous obstruction probably caused by operation previously, opening lateral sinus and from packing to central hemorrhage. Aseptic clot would follow.

Ritter, J.—"Uber Gunstigen Ausgang Wahrscheinlicher Thrombose des Sinus Cavernous Otitischen Ursprungs." Extract translated from Zeit. f. Hals-Ohr.-Nasenheilk., 1, 1922, 352.
Hölscher collected up to 1904, 10 cases in which a disease diagnosed as otitic thrombosis of the cavernous sinus recovered. Of these ten cases the following five, he thinks, lack definite proof: Wreden, Styx, Hoffman, Keller, Bezold. The author abstracts the five proven cases of Hölscher, Bircher, Tervaer, Preysing, Kolb and Brieger, and the reported cases of Johnson, Boonacker, and Bourgeois, and reports one of his own from Boeninghaus' clinic. Ritter's case was certainly an aseptic clot, as it was without fever. Tervaert's was a recovery from secondary abscess in the orbit.

Uchermann, V.—Phlebitis of the cavernous sinus without thrombosis.
Monatschr. f. Ohrenheilk., 522, 1921, 1710.
Ear. Several weeks. Classical symptoms. Recovery. Probably came from throat with local meningitis. The ear probably had nothing to do with it.

Wreden.—St. Petersburg Zeitschr., 1869, p. 98.
Arch. Ophth. and Otol., 1874, Vol. IV, p. 52. Abstracted by Coupland, Tr. Op. Soc., 1888.
From the ear through right lateral sinus to right jugular

and superior longitudinal, and to left jugular and left lateral sinus. Facial erysipelas and thrombosis of right cavernous. (Doubtful.)

Wrigley.—Jour. Laryngology., Aug., 1921, p. 381.
Probable cavernous sinus thrombosis. Nystagmus. Proptosis. Edema. Recovery.

## BIBLIOGRAPHY OF REPORTED RECOVERIES—NOT AURAL

Chisholm and Watkins.—Twelve cases of thrombosis of the cavernous sinus.
Arch. of Surg., Nov., 1920, Vol. 1, pp. 403-512.
Case 8: Recovery following administration of stock and autogenous vaccine, and drainage of metastatic osteomyelitis.

Cushing.—Treatment of major neuralgia of the trigeminal nerve.
J. A. M. A., 1905, XLIV:773, 860, 920, 1002, 1088.
Aseptic thrombosis, from Gasserian operation.

Gilberti.—Policlinico (Sez. Chir.), Vol. 26:268, Rome, 1919.
Recovery with eye symptoms.

Dwight and Germain.—(Bost. Med. Jour., 1902), citing 14 cases of spontaneous recovery. In 134 autopsies showed 31 thromboses limited to one sinus. 65 had both affected, and 28 other sinus.

Knapp and Hartley.—Arch. of Ophth., 1900, p. 77. Tumor case.

Landsberg.—Centralbl. f. Augenheilk., 1883, p. 129, abstracted by Complin, Tr. Ophth. Soc. of U. K., 1888.
It is certainly not verified that it was not simply an orbital infiltration.

Mills, C. K.—Philadelphia Hospital Reprints, 1890, p. 261.
Unilateral ophthalmoplegia, probably dependent upon thrombosis of the cavernous sinus, associated with basic meningitis. Not verified as infective.

Payr.—Münch. Med. Wchnschr., Vol. 62, p. 987.
Body infection. Arm. Classical. Recovery.

Seggel.—Klin. Monatsbl., Aug.-Sept., 1907; Ophthl., 1909, p. 188.
Patient recovered with great limitation of the field, pointing toward disease of chiasm of the optic tract.

Stocker, F.—Rare case of thrombosis of the cavernous sinus following orbital thrombophlebitis. Arch. Ophthal., Vol. 35, 1906, p. 373.
Traumatic. Recovery with atrophy of the nerve.

Zentmayer and Weisenberg.—N. Y. M. J., 83:184, 1906.
A case of acute retrobulbar neuritis, probably due to sinusitis. Recovery. No vision.

# CHAPTER XI

## EMBRYOLOGY

Embryologically the cavernous sinus is somewhat different from the other venous trunks of the head, which are formed by splitting of the dural layers by endothelial ingrowth.[1] On the other hand, the lateral wall of the cavernous sinus is made up of two layers, a thin deep layer continuous with the sheath of the carotid artery and a thicker superficial layer from the dura of the middle fossa. Between these two layers the nerves pass (Ferron),[2] carrying with them prolongations of the dura as they pierce it.

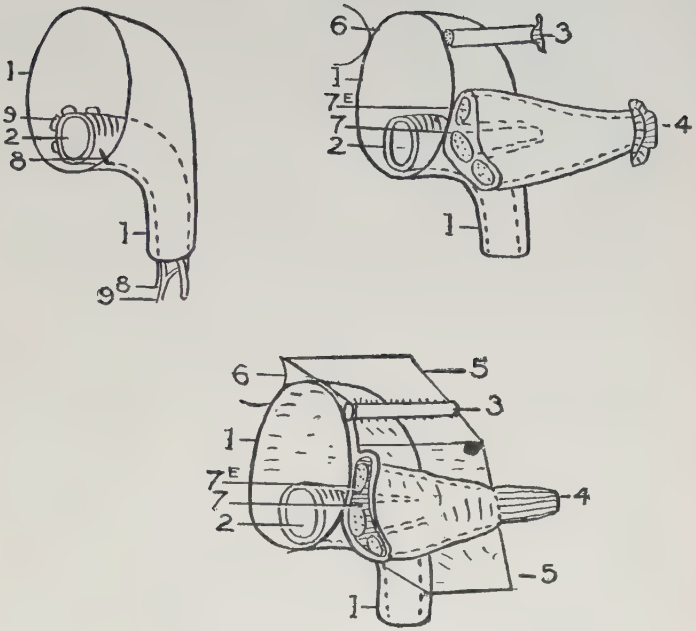
Lange [3] divides the cavernous sinus into two parts; a ventricular superficial cavity containing the nerves, and a deeper more spacious one nearer the bone, for the blood.

The 3rd, 4th, 6th and first division of the 5th nerves are actually in the sinus. The first three (3rd, 4th and 6th) having in reality left the encephalic cavity when they enter the sinus, are surrounded by the usual arachnoid prolongations common to all cranial nerves as they leave the skull, but differ from the other nerves

[1] Whitnall, S. E.: Quoting Langer—*Anatomy of the Human Orbit,* London, p. 318.

[2] Ferron, M.: "Note sur constitution des parois du sinus caverneux," *Jour. de Medécine de Bordeaux.*

[3] Lange, V.: "Cavernous Sinus," *Sitzungsberich der Kaiserlichen Akad. der Wissenschaften,* 1885, p. 320.




1. Carotid sheath. 2. Internal carotid artery. 3. Sympathetic nerve in the dural sheath. 4. Trifacial nerve in the dural sheath. 5. Thick superficial layer of the dura mater. 6. Dura mater of the sella turcica. 7. Superficial lamina, and 7E deep lamina of Meckel's cavity. 8. Nerve fibers. 9. Small veins of the venous plexus. ("Note sur la constitution des parois du sinus caverneux et les rapports des nerfs oculomoteurs et trijumeau avec ses parois externe et superieure."—Par le Dr. Michel Ferron. *Journal de Medecine de Bordeaux.* 1913, Feb. 9. No. 6.)

in that they enter a venous tract instead of a specific mucous membrane, as does the olfactory or the soft parts, as do the 9th, 11th and 12th, or extend to a ganglionic prolongation of the brain, as do the 2nd, 5th, 7th and 8th.

The cavernous sinus, like all the venous trunks of the body, presents frequent anomalies of position and communication.

Occasionally one side is found to be absent, in which case phlebitis, even without occlusion of the remaining side, would immediately be followed by a unilateral ocular edema and exophthalmos. The embryonic petrosquamous sinus may be patent, emptying directly into the cavernous sinus—as in a case reported by Vernieuwe,[4] in which thrombophlebitis of this anomaly caused temporal edema and orbital symptoms without lateral sinus involvement.

[4] Vernieuwe: Anomalies of the sinuses of the dura mater, "their clinical significance in otology," *Rev. de. Laryngol. d'Otol. et de Rhinol.* 42:239, Paris, May 15, 1921, and 42:207, April 30, 1921. Abstracted in the *Internat. Medical and Surg. Survey,* June, 1921, pp. 172 and 174. Good bibliography.

# CHAPTER XII

## SURGICAL ANATOMY AND PATHOLOGY

### SURGICAL ANATOMY

On account of its median position within the cranial cavity and its intimate relationship with the internal carotid artery and nerves (3rd, 4th, 6th and ophthalmic division of the 5th), the cavernous sinus can only be approached surgically either through the orbit, in front; by way of the petrous pyramid from behind; laterally along the floor of the middle fossa; or mesially through the sphenoid sinus.

Again, the sponge-like structure of the cavernous sinus will not permit of free surgical drainage no matter what route is selected; for, anatomically, it is impossible to remove pus and infected clots completely as can be done in the more favorably situated tubular pathways of the sigmoid and transverse sinuses. Such being the case, if infection within the sinus is to be combatted, not only must the sinus be exposed before the infection has spread throughout its many ramifications, but the limited area of the operative opening demands an application of the knowledge of minute surgical pathology of the cerebral venous trunks, which has now become very exact because of the large number of operations that have been conducted on

the lateral and sigmoid sinuses. A detailed consideration of the pathology of thrombophlebitis, in general, is thus necessary for successful surgical treatment.

**Principles of Applied Surgical Pathology in Cerebral Sinus Infections.**—The initial pathological process present will depend upon whether:

(1) the virulence of the infection; or
(2) the protective reaction, *viz.*, the localizing clot is playing the more prominent part.

**Types of Lesions Recognizable Clinically or at Operation in Cerebral Venous Sinus Involvement.**—

(a) Septic vegetative vasculitis (only seen at post-mortem;
(b) Parietal thrombophlebitis (mural clot); an early stage of
(c) Localized obliterating thrombophlebitis; which frequently is followed by
(d) Gangrenous thrombophlebitis;
(e) Fulminating phlebitis,

all of them are different degrees of septic invasion of a venous sinus itself; in contradistinction to the

(f) Aseptic (protective compensatory) thrombosis.

Each of these types demands a distinct surgical recognition as each presents different clinical manifestations and technical difficulties to be overcome.

## SURGICAL PATHOLOGY (GENERAL CONSIDERATIONS)

In view of the condition of the clot as found at post-mortem in the cases herein reported, and during operation in a large number of cases of the lateral and transverse sinus diseases, I am of the opinion that

the current conception of thrombosis of the cerebral sinuses—as fundamentally a septic clot occluding a large venous pathway—should be somewhat revised. For in the slow compensatory cases, the thrombus forms on the walls and in the caverns of the sinus and frequently entirely obliterates it before the clot itself is infected. The clot is primarily (Case 15) protective in origin.[1] Later, however, if the protective obliterating clot does not succeed in limiting the infection to the vasa vasorum, to the wall of the sinus, or the cavity of the sinus, the clot itself may become purulent with the discharge of micro-organisms into the blood stream thus becoming a general blood stream infection.

Again, the extension of the clot is largely *due to the mechanical* slowing of the blood current, and is nature's further effort to strengthen the barrier against entrance of the infection into the blood stream itself; as, even when the clot is very extensive, both ends of an obliterating thrombus are frequently entirely aseptic.

In many cases this slow compensatory, aseptic thrombotic clotting is the outstanding process through the whole period of the disease.[2] The protecting clot (involving many venous sinuses and extending great distances, sometimes with multiple areas of purulent disintegration separated by areas of aseptic sinus

[1] Rodger, T. Richie: *Jour. Laryn. and Otol.*, 1921, Vol. 36, p. 169—Case 2; "Posterior part of sinus showed non-infected clot."

[2] Kolb's and Ritter's cases, which recovered, had symptoms of cavernous sinus obstruction for three months without any elevation of temperature.

obliteration),[3] demonstrates nature's effort to limit the infection of the already invaded and now purulent clot;[4] [5] while many of the cases of thrombophlebitis, which in the beginning are infected, go on to complete obliteration of the blood stream without suppuration, as in the following case.

CASE G.

*Non-infective obstruction of ophthalmic artery or vein from furuncle of nose. Spontaneous recovery.*

Seen in consultation with Dr. R. C. Dodd.

*History:* The patient had had a furuncle in left nostril, which was twice incised. Followed by severe illness, headache, chills and fever associated with swelling on the right side of face. Right eyelid being greatly swollen; left eye moderately so, but only for a few days. Diagnosed as facial erysipelas.

*Three Weeks Later:* (Dr. Dodd), marked swelling and edema of right eyeball with proptosis and chemosis of conjunctiva. Palpation of orbit disclosed an indistinct mass at the inner corner of the right eye, but an incision into it failed to reveal pus.

*Examination:* (25 days after onset of illness) proptosis of right eyeball, with chemosis of the ocular conjunctiva below; and dilatation, especially of the superior conjunctival veins. Pupil dilated and does not react to light. Fundus: retina whitish; slight indistinctness at the margin

[3] Riviere, A., and Bartoin, R.: "Phlebitis of Longitudinal Sinus," *Lyon Chirurgical,* 19; 6, 1922, p. 679, record a chronic case lasting six months with involvement of all the venous sinuses of the *head* and internal jugular veins from a double otitis media.

[4] Coupland: "Left sinus (original side of infection) occluded by non-purulent clot."

[5] Esch, A.: "Pathologic Anatomical Changes of the Venous Sinuses in Man, in Otogenic General Infection." Part I. *Zeitschrift f. Hals—Nasen—u. Ohrenheilkunde,* Vol. IX, p. 46, 1924.

of the disc; retinal veins rather full, arteries small. Right vision, negative. Vision 20/30. Infection of sinuses excluded.

*Subsequent History:* The eyeball slowly returned to normal condition. Fundus: secondary atrophy of the nerve.

*Diagnosis:* Obstruction to blood supply of orbital arteries and veins from facial infection; or possibly a cured cavernous sinus thrombosis, the retrograde thrombophlebitis having travelled upward from the nose to the ophthalmic vein, and possibly the artery.

**Elimination of Micro-organisms from Blood Stream.**—The experiments of Teale [6] demonstrate that the blood stream is freed from circulating micro-organisms by the bacteria being caught in the capillaries of the spleen, kidneys, lungs, and liver by a process of local clotting of blood. The clots which contain micro-organisms, acting as irritants are attacked and destroyed by the phagocytes of the blood and the tissues.

In overwhelming infection of the blood stream, one of the uniform pathological findings is the presence of infarcts and abscesses in the spleen, kidneys, lungs and liver, and one reason why the patient is cyanosed is because of the clotting—which occurs in the capillaries of the lungs in nature's attempt at eradicating the bacteria by phagocytosis.

Case 21.

Patient of Dr. E. W. Erler.

*Previous History:* Has had several infections around head during past three years: (a) iridocyclitis with secondary cataract, left; (b) blennorrhea lacrimal sac, right; (c) chronic antrum disease with acute exacerbation, right.

[6] Teale, F. H.: "Bacterial Intoxication," Horace Dobell Lecture, *Lancet*, August 7, 1920, p. 279.

*Present Illness:* Eight days ago had a pimple on the left side of the lip followed by high fever and swelling of the left side of the face. "Lip incised under cocaine on the second day, again on the third, and under ether on the fifth day of the disease." (Staphylococcus aureus.)

Last two days patient has been semi-conscious, high temperature, with rigidity of neck. Cough—respiration 40 to 50; respiratory grunt—Cheyne-Stokes respiration (septic pneumonia). Leucocytes 9,000. The swelling of the face and lids has somewhat subsided, but the eyelids are bluish, and there is chemosis and exophthalmos of both eyes, chiefly of the left. Fundus: slight papilledema, with greatly enlarged veins.

Death in coma on eighth day of infection.

Whether the bacteria themselves or their toxins by injury to the intima of the vessel walls or invasion of the vasa vasorum, are the immediate cause of the liberation of thrombokinase (or an allied substance from the tissues) is not known; but the clotting and slowing of the blood current—thrombosis—is one of the protective reactions of nature; a reactive process present in or surrounding *all bacterial invasions.*[7]

A thrombus thus produced should therefore be regarded as nature's protective effort to prevent invasion of the blood stream, not, as is the usual view, that

[7] This capillary clotting must be caused by the liberation of thrombokinase or thrombin, in stagnant blood—as neither venous stasis alone nor the presence of thrombin or thrombokinase in moving blood causes clotting. The change which takes place to produce clotting might be expressed as follows: "Thrombinogen plus a calcium ion (both normally present in the blood), plus thrombokinase (from the tissues), equals thrombin; which latter (thrombin), plus fibrinogen (present in the blood) produces a fibrin clot." Collingwood: "Embolism and Thrombosis," *Proceedings of Royal Society of Medicine, Sec. of Surgery,* 1921-22, p. 29.

the thrombus is necessarily the agent of bacterial dissemination, something that must be totally eliminated surgically lest the infection should spread. This latter is only correct if the clot itself has become infected, when it may be either purulent or putriform, rapidly progressing or not.

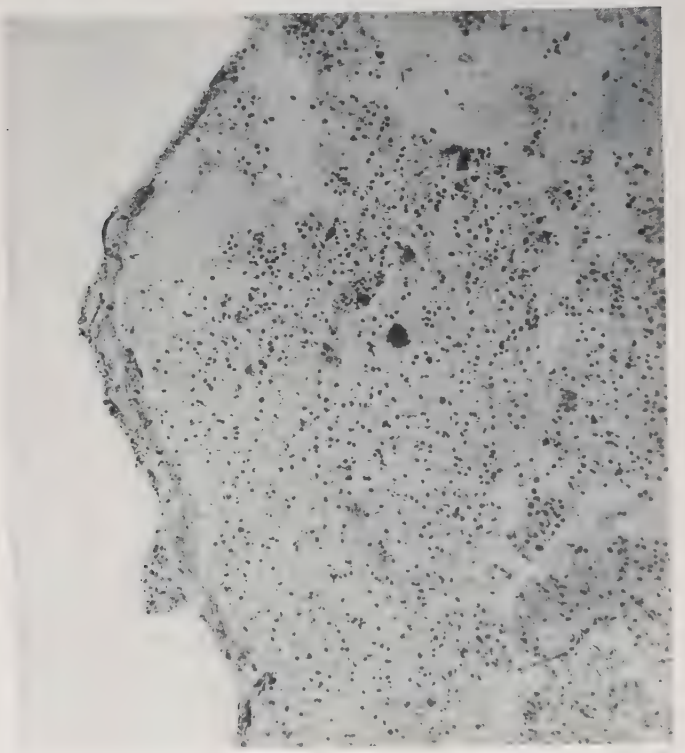
When the clot is infected, it is generally necessary to eliminate it surgically; but prior to actual infection the protection of the clot and its utilization to limit the infective process offer the chief hope of the patient's recovery in cases of cavernous disease. (See illustrations.)

In a similar manner, in the different forms of basal sinus infections the surgeon should regard a parietal thrombus as nature's way to prevent the invasion of the blood vessels by the micro-organism; and vegetative thrombosis as nature's attempt to encapsulate the micro-organism which has already gained entrance.

**Non-Infective Thrombosis.**—A thrombus from infection differs from the slowly growing non-infective variety (as seen in venous occlusion in different parts of the body, such as the femorals) both microscopically and mechanically.

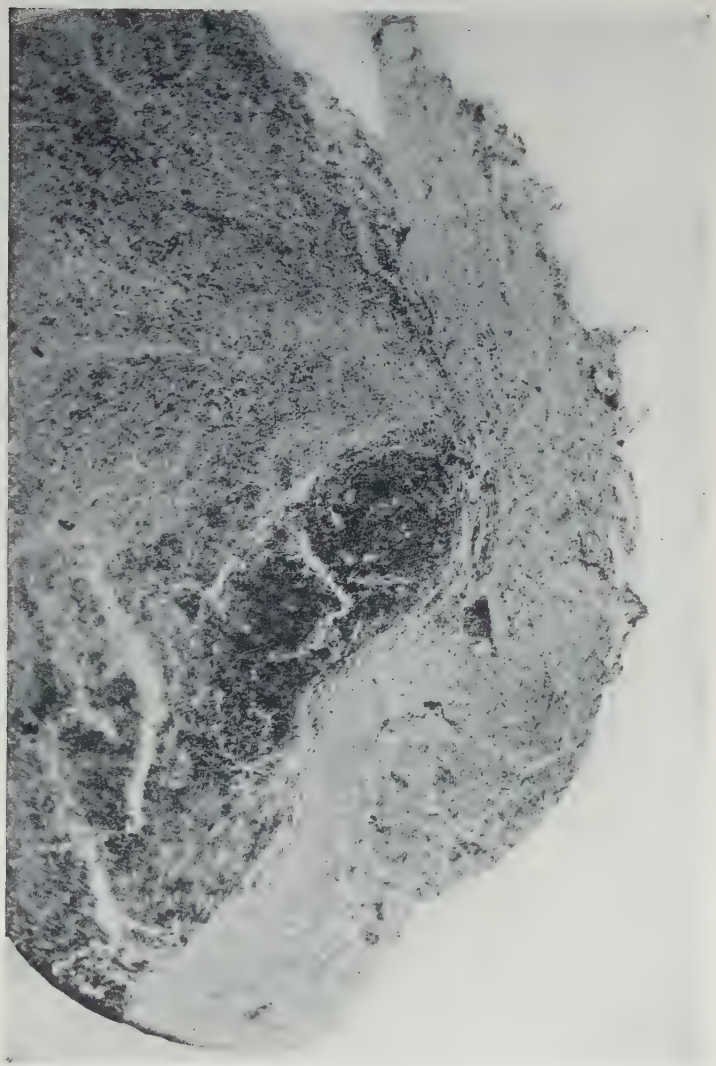
A non-infective thrombus is of blood-plaque origin with a later addition of white and red cells held together by a network of fibrils from the blood-plaque. There is no fibrin formation in the basal part of the thrombus. This type of autochthonous thrombus originates from an agglutination of the plaques to the inner wall in the presence of moving blood; usually, but not necessarily, from injury to the wall from some chemicophysical agent. This is the "white" portion of the thrombus. The "mixed" portion is a subsequent addition and is due to the deposit of blood

Vein from Acute Case Dying on Third Day of Disease.



Ophthalmic Vein.—*Suppurative Thrombophlebitis.* The outer coat of the vein shows a leucocyte exudate, the cells being chiefly polymorphonuclear variety. The vessel wall appears to be normal. In the lumen of the vessel are colonies of bacteria (on culture found to be the staphylococcus pyogenes aureus). The cells are polymorphonuclear leucocytes.

Vein from Patient Suffering from Cavernous Sinus Thrombophlebitis, Who Lived a Few Weeks After the Beginning of Sepsis.



Thrombosed Vein.—Section shows fairly recent thrombosis at one part commencing organization. At this point the vessel wall is greatly thickened and shows small hemorrhagic areas and the formation of young capillaries. The clot shows no signs of septic breaking down.

cells in the "eddy" occasioned by the slowing of the blood current. (Aschoff.[8]) On the other hand, in infective thrombosis the microscopic picture is that of blood clotting; the clot being held together by the usual fibrin formation.[9]

As a result of post-mortem observations (Case 16) and an examination of reports in this literature I am persuaded that local thrombosis of the cavernous sinus and of the lateral sinus—aseptic and protective in nature—is a very frequent occurrence.

It is only when the infection extends and invades the clot itself that general infections of the blood stream occur. (This is certainly the usual process when the cavernous sinus is attacked from the rear as in petrosal cases.)

### CLINICAL TYPES OF SINUS INVOLVEMENT

(a) **Septic Vegetative Vasculitis.**—(Septic vegetation on the sinus walls) is the result of irritation from micro-organisms which have become encapsulated on the sinus wall, the excrescences thus formed having successfully localized the micro-organisms to one place. Undoubtedly this is a part of the protective process whereby septicemia is cured.

Impressed by the accidental finding of septic vegetative vasculitis in a case of superior longitudinal sinus[10] (also Cases 16 and 20), the writer instituted a survey of the

[8] Aschoff, Ludwig: "Thrombosis." Cartwright Lectures for 1913. *Arch. of Internat. Med.*, Vol. 12, No. 5, November, 1913, pp. 503-525.

[9] Haward, Warrington, F.R.C.S. Eng.: "Phlebitis and Thrombosis." The Hunterian Lectures. Delivered before the Royal College of Surgeons of England, in March, 1906. William Wood & Sons.

[10] Eagleton, W. P.: *Brain Abscess, Its Surgical Pathology and Operative Technic.* Macmillan, 1922, Case 15, p. 71.

literature concerning the subject and found but few references to such a condition. However, the references are sufficient in number to warrant the statement that this condition is probably far from rare, but that it is not generally recognized because the lumina of the cranial sinuses have not been systematically examined at a distance from the primary focus of infection.

*Perivasculitis, Invasion of the Vasa Vasorum.*[11]— In perivasculitis the outer fibrinous coat of the sinus is first attacked by micro-organisms from an adjacent bony caries, or a perisinuous abscess. The inflammation thus originates a protective reaction within the sinus itself; and there is at first formed a parietal thrombosis in an effort to prevent the invasion of the endothelial lining of the sinus and of the blood stream itself. (See microphotograph on page 125.)

(b) **In Parietal Thrombophlebitis,** the mural clot involves only the osseous wall of the sinus. It is at first a protective process due to nature's effort to prevent infection of the sinus itself. It is a frequent condition. The majority of cases of thrombophlebitis do not pass this stage if the adjacent area of bony caries is promptly removed. When the mural clot becomes infected before the formation of an occluding thrombus, some constitutional symptoms of blood stream infections are present—high temperature continuous, or pyemic; and leucocytosis with frequently positive blood culture.

These cases of parietal mural clot infection from an adjacent bony caries are very common in the lateral

[11] Behm, M.: "Zur Pathogenese und Klinik der primaren otogenen Thrombose des Bulbus venae jugularis," *Zeitschrift für Hals-Nasen- und Ohrenheilkunde,* 4:394-404, 1922-1923. (Bibliography.)

sinus; and when early and properly treated result in a high rate of recoveries. In my experience, they are the most successful and satisfactory cases to treat surgically. Operation immediately converts a general blood stream infection, fed from a suppurative focus within the sinus, into a localized septic infective process. The pyemic remissions of temperature give place to a lowered but still elevated and continuous one, which disappears within a few days.

It is well known that the larger the sinus the less apt it is to be the site of a thrombophlebitis, and, conversely, the smaller the sinus the more apt it is to be involved and obliterated. It is in the large lateral sinuses filled with blood that the treatment of the sinus itself and the elimination of infection by incision of the mural clot are attended with the most striking results.

This type of mural or partially obliterating clot frequently occurs in the cavernous sinus, especially when the original focus is supplied from a sphenoidal suppuration (Case 16), or originates from the cells at the apex of the petrous pyramid. (See Case F.)

In a partial (mural) clot the free opening of the sinus and its complete obliteration by pressure to control hemorrhage may favor an extension of the infective phlebitis.

(c) **Localized Obliterating Thrombophlebitis,** *in which, due to infection of the vasa vasorum or intima, an obliterating clot is formed, which may or may not break down into pus.* It is by an extension of this process that the opposite cavernous sinus is primarily occluded even when the infection is definitely in the

cavernous sinus; as post-mortem and microscopical examinations demonstrate that, although both cavernous sinuses may be occluded by clot, the side primarily attacked may be the only one actually invaded by bacteria. (Case 15.) The aseptic protective clot of the opposite sinus may cause enough vascular disturbance to produce ocular symptoms, and yet it need not be removed if the extension of invasion by bacteria from the opposite sinus can be prevented.

The slowing of the blood current, so necessary for the formation of a thrombus, finds in the jugular bulb and in the many septa of the cavernous sinus a most favorable site for its development, providing the blood itself is altered by bacteria or toxins attacking its wall.

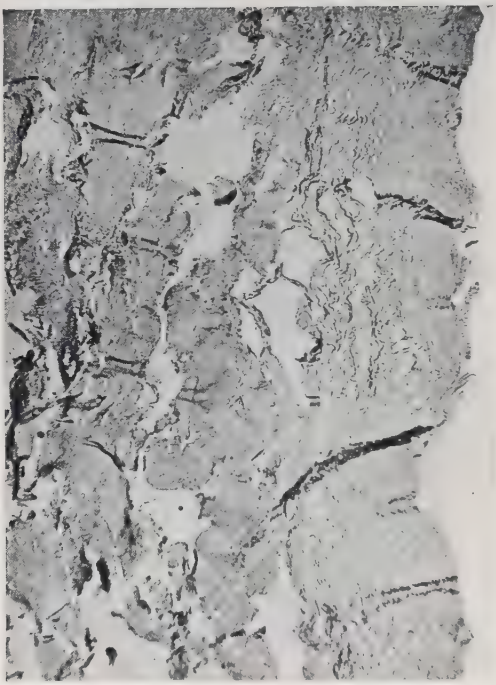
**External Appearance of Sinus.**—When the mural or obliterating protective thrombus has become infected by bacteria, the outer sinus wall (because of its fibrinous character) may show no sign of disease, as the intima is the structure of the sinus chiefly involved.

In a jugular case the writer, on opening the neck, exposed an apparently large jugular vein, the walls of which were not matted to the adjacent tissue. When opened, it was found to be filled with pus, the localized pus cavity within the vein extending from the jugular bulb to below the clavicle.[12]

In Case F, free pus extended through the whole length of the jugular vein but was unsuspected during life, there never being any complaint of pain or tenderness.

In Case 17, the absence of pain and tenderness (although free pus extended from the middle of the neck up into the

[12] Eagleton: "Vestibular Tests in Intracranial Surgery." Case 4. Tr. Amer. Laryngol. Rhinol. and Otol. Soc., 1922.



Jugular vein at operation was apparently normal and contained circulating blood. Vein excised between two sutures. Culture from blood in excised section gave streptococcus hemolyticus, 15 colonies to cc. Microscopic section shows intense hemorrhagic periphlebitis and thrombosis of the small vasa vasorum. (A. J. Eagleton.) (Case of Dr. H. C. Barkhorn.)

sinus at the knee) delayed operative interference for at least a week.

In Case 15, the post-mortem suggested that, if instead of stopping after performing a Grunert's operation on the jugular bulb, the common carotid had been ligated and the cavernous sinus opened, the patient's life might have been saved.

(d) **Gangrenous Thrombophlebitis** comprises the most severe cases, in which the sinus becomes gangrenous, the lumen being rapidly obliterated and often resulting in necrosis of the sinus and extravascular abscess formation—a condition frequently encountered at operation on the lateral sinus.

Although so fatal, it is fundamentally largely a protective process; nature trying to convert a virulent vascular invasion into a local process, cut off from the blood stream. When such a process attacks the cavernous sinus, meningitis rapidly supervenes.

(e) **Fulminating Phlebitis.**—In this, the rapid and virulent extension of the infection causes a profound blood sepsis with little or no thrombotic formation—the infection extending great distances in the vasa vasorum of the veins. Excision is the only hope of surgical relief.[13]

In a case of jugular vein excision (Dr. Barkhorn) the apparently normal vein was filled with blood—cultures from which revealed streptococci. The vasa vasorum of the jugular were occluded by thrombi and invaded by bacteria. (Dr. Arthur Eagleton.) See illustration.

[13] Grueter, H. A.: "Die Wurzelgebiete der Hirnsinusthrombosen," *Virchow's Archiv für Pathologische Anatomie u. Physiologie*, 247: 411-420 (1923-1924).

# CHAPTER XIII

## OPERATIVE TREATMENT

To meet with success the surgeon must be prepared to act on the early appearance of symptoms—he must act with faith in his own convictions instead of waiting for developments. He must have a definite operative policy, although the diversity of treatments advocated would suggest that chaos, as to any such policy, now exists.

**Operative Policy.**—This must be planned to combat pathological conditions: (1) as to the type of infection —whether it is (a) an acute infective gangrenous thrombophlebitis (in which the septic process is rapidly spreading, the toxemia is profound, and the blood infection severe—here surgery to the sinus itself offers little prospect of recovery), or (b) a slowly obliterating protective thrombophlebitis (in which, although the process is primarily infective, the thrombus itself is protective; the course is slow, and the blood stream infection and toxemia not severe; these hold out a fair chance for recovery by surgical intervention). (2) The operative attack must follow the route by which the infection entered the sinus. In addition, the operation itself must be (3) carried out before the cavernous sinus as a whole has become infected. The diagnosis must be made before the accomplishment of the

fact; in other words, the disease must be attacked before the sinus is the actual site of a suppurating clot.

**Technic: Mental Attitude of Surgeon.**—As discussed in the section on pathology, it is of importance that the surgeon should mentally discard the term sinus thrombosis, which implies that the clot is the object of the surgical attack. The thrombus in itself is a protective process. He should think of a *septic phlebitis of a large venous radicle;* and while in the attempted control of the process it may be desirable to remove the clot when infected, or drain it when purulent, the infective process itself can only be limited by clot formation. Consequently, the surgeon's effort should be to encourage and assist this formation of an aseptic thrombus.

**Surgical Indications.**—In an effort to control a septic phlebitis of the cavernous sinus the surgical indications are (1) eradication of the original focus of infection; (2) as free drainage as is possible through the path of the original infection, and (3) to place the inflamed and thrombosed sinus at rest by ligation of the internal or common carotid artery of the affected side.

**Placing the Thrombosed Sinus at Rest.**—It is well known that for the cure of phlebitis (be it of the hand, leg or other soft part) absolute rest is necessary, and that any surrounding muscular action (such as walking in thrombosis of the leg) causes an extension of the phlebitis. Rest, however, in the case of an involvement of the cavernous sinus is impossible because of the constant impact of the inclosed internal

carotid artery as it courses in the lumen of the sinus itself.

This continuous arterial pulsation giving rise to incessant movement of the inflamed venous radicle must cause an extension, not only in the wall of the affected cavernous sinus, but also into the adjacent sinuses—the intercommunicating—even though the original cavernous sinus is drained.

To my mind, it is this pulsation of the internal carotid that has been the chief factor in the almost uniform extension of the infection, as the arterial movement prevents a limitation of the infective process, even when the affected side is drained. Prior to the first report of the case (Case 22), apparently no one had realized the importance of this anatomical relationship.

The preliminary ligation of the common carotid not only furnishes rest to the parts, but renders negligible operative hemorrhage (Cases 22 and 23); this otherwise is apt to be excessive because conducted through the inflamed congested tissues of the orbit or petrous pyramid. (Cases 10 and 14.)

Tying of the internal carotid is just as efficacious in furnishing rest to the inflamed structures as ligation of the common, but it does not control surface hemorrhage as well and takes longer to perform.

In spite of its supposed dangers, I have rarely seen untoward effects from ligation of the common carotid in a moderately large number of cases, except cases J, K and M in all of which other factors entered.

## INFLUENCE OF DIMINISHING THE BLOOD SUPPLY IN OCULAR VENOUS STASIS IN CASES OF TRAUMATIC ARTERIO-VENOUS ANEURISM BY LIGATION OF THE ARTERIAL SUPPLY IN THE NECK.

While the origin of the exophthalmos, chemosis, and swelling of the lids in cases of traumatic pulsating exophthalmos is largely due to the fact that the venous channels, acting as incoming arterial pathways, increase the amount of blood in the orbit; the real cause of the proptosis is the induced venous stasis, and to this extent it is somewhat similar to the exophthalmos for cavernous sinus thrombophlebitis. My experience with traumatic venous aneurism is largely responsible for my adopting ligation of the common or internal carotid at the time of the jugular resection in those cases of cerebral sinus thrombophlebitis in which, at the time of the operation, there may be doubt of the infection being fully controlled by drainage, either because of the position of the infection (such as jugular bulb and suspected petrosal cases), or because of its virulence. The results obtained have fully warranted my expectations, and I advocate the procedure in all similar cases.

The chief surgical value of the following cases, so far as cavernous sinus phlebitis is concerned, lies in their demonstrating the control of abnormal ocular venous stasis (manifested by exophthalmos, chemosis, intraocular hemorrhages and fundus changes) when the arterial or venous (Case E) blood supply is diminished.

In venous arterial aneurism following rupture of the internal carotid artery into the cavernous sinus,

the exophthalmos is controlled by ligation of the common carotid in the majority of cases.

Case H.

*Exophthalmos, chemosis and intra-ocular hemorrhages of unknown origin; possibly from spontaneous arteriovenous communication. Ligation of common carotid. Recovery.*

Patient of Drs. R. C. Dodd and W. R. Broughton, Mountainside Hospital. Woman's age, 50 years.

*History:* Good health until April, when she had violent pain in head with vomiting; followed next day by diplopia with drooping eyelid from paralysis of left third, associated with noise in her ear. O. E. negative; sight normal.

Gradual subsidence of all symptoms; 7 weeks later none present. No more headaches.

In September, noticed beginning dilatation of the veins of the conjunctiva and a continuous noise in head which had recurred off and on during the summer. The conjunctival condition gradually increased, and in November there developed a slight exophthalmos.

*January 8:* Suddenly awakened in the morning with a feeling of stiffness over the eye and greatly reduced vision. Rapid development of chemosis and increase of exophthalmos. "Globe deeply injected from venous stasis."

*Fundus:* Marked venous engorgement and varicosity, no hemorrhages.

Disappearance of noise in head and deafness since beginning of this attack. Heart normal; B. P. 140. Urine, negative. Five days later, fundus: multiple hemorrhages; vision, ability to count fingers. X-ray: "shows a defined mass in the apex of the orbit; the outer side of the sphenoidal fissure seems to be more or less obliterated."

*Diagnosis:* "It must be a vascular lesion because of the sudden onset; probably a venous arterial aneurism, which caused the first vascular breakdown in April, and which has gradually leaked through the whole summer, causing

Case H.—Exophthalmos of vascular origin.

the venous engorgement of the ocular conjunctiva, and recurrence of noise in head in September, and the exophthalmos in December. The venous arterial communication probably tore wide open in January, causing the venous blocking as the bruit of arterio-venous aneurism is only present when the opening between the vessels is small. If the tear in the abnormal channel becomes large, it may disappear; while the signs of venous stasis greatly increase. Of course, this vascular disturbance may be secondary to a tumor."

*January 30, Operation:* Ligation of left common carotid artery with strip of fascia lata. Rapid disappearance of exophthalmos, chemosis and intra-ocular hemorrhages.

*May 7:* "All structures normal except a few varicose veins of conjunctiva and a residuary paralysis of the inferior oblique, which causes a slight head tilt. Vision, normal."

Case I.

*Arteriovenous aneurism from fracture of skull. Ligation of both common carotids. Partial recession.*

Patient of Dr. W. Doremus.

*History, October 26:* Admitted to Stumpf Memorial Hospital, suffering from a fracture of base of skull with bleeding from left ear. Coma alternating with delirium, followed by headache and dizziness.

*Examination:* Right exophthalmos and chemosis, paralysis of external rectus. Spontaneous nystagmus. Fundus; dilated veins. Bruit over right side of head. Left eye blind from old cataract.

*Diagnosis:* Fracture through left petrous and right orbital apex, causing arteriovenous aneurism.

Exophthalmos and chemosis gradually subsided; but two months later, following a cold, both increased markedly so that cornea began to ulcerate.

*January 29, First Operation:* Ligation of common carotid with fascia lata. Followed by reappearance of bruit

3 days later. Religated, with disappearance of bruit and incomplete recession of exophthalmos.

*One Year Later:* Has been in good health and has attended to his work, but during past two months there has been recurrence of exophthalmos and there is now ulcer of cornea. Bruit present on auscultation. Enormously dilated supraorbital and angular veins.

*Operation:* Large supraorbital vein ligated, just inside of orbital margin. Followed by increase of exophthalmos and spread of corneal ulcer.

Division of external angular ligament failed to allow cornea to be covered by lids.

Few days later severe spontaneous hemorrhage from operative wound; controlled by firm packing. After recurrence of spontaneous hemorrhage.

*Operation:* Ligation of *opposite* common carotid. Followed by incomplete recession of exophthalmos and healing of ulcer.

## DISTURBANCE OF CEREBRAL CIRCULATION FOLLOWING ARTERIAL LIGATION TO PLACE PARTS AT REST.

The symptoms of disturbance of the cerebral circulation from surgical obliteration of the common or the internal carotid may result from:

(1) Nutritional death of the cerebral tissue because of lack of sufficient blood to a limited area of the brain. This variety is rare and occurs immediately after the ligation. It is manifested by a hemiplegia of the opposite side; usually from anemia of the region supplied by the middle cerebral (the most important of the terminal arteries of the internal carotid). It is apt to occur in patients suffering from chronic arterial disease, such as diabetes, etc. (Kadish and Peterson.) It may be avoided by the temporary applica-

Case I.—Exophthalmos from arteriovenous aneurism following fracture of skull.

tion of Crile's arterial clamp at the beginning of the operation; to be followed, if no symptoms of vascular cerebral disturbance have meanwhile appeared, by a fascia lata ligature.

Case J.

*Death preceded by hemiplegia from nutritional death of cerebral tissue, following ligation of the internal carotid performed on a diabetic patient suffering from chronic thrombophlebitis of the jugular bulb.*

*Synopsis of Symptoms:* Has had diabetes for years and taken insulin treatment during past year.

History of pain in right side of head with earache during past seven weeks. Pain relieved temporarily by paracentesis. Slight discharge, which contained streptococcus nonhemolyticus. Rapid healing of drum but continued right-sided cephalalgia; the pain occurring in attacks associated with pain behind right eye and in occipital region. Several chills. Very slight fever and irregular sweating at night.

*Examination:* Slight fullness in right side of neck. Is tender over the posterior portion of the mastoid (Griesinger's sign of phlebitis of mastoid emissary). Drum membrane normal but lusterless. X-ray shows mastoid cellular cloudiness. O. E. vessels small (such as seen in arterial disease).

Blood count: erythrocytes 4,260,000, leukocytes 7,400, polynuclears 78 per cent., lymphocytes 22 per cent. Culture, negative. Urine: sugar + + + +.

*Diagnosis:* Thrombophlebitis of mastoid accessory vein secondary to lateral sinus thrombophlebitis. (It should have been added, in a *patient suffering from chronic arterial disease,* the result of diabetes.)

*Operation* (local anesthesia): Mastoid and antrum free from disease. Exposing the deep cells over the cerebellum disclosed a diffuse mass of firm granulated tissue; which did not look infected. Sinus opened low down, liberating thin grumous pus. Wide incision of sinus

revealed a mass just above the bulb with what looked like adhesions running from the anterior to the posterior wall, and a thick exudate lining all surfaces giving the impression of an old encapsulated cicatrizing abscess. No bleeding obtained from below. Incision upward toward the knee revealed clot with an area of free pus. Free bleeding obtained at a distance of about 1½ inches behind the knee.

Internal jugular ligated at junction of facial, above which the jugular was very small. (Evidently the phlebitis had extended as far as where the facial veins began.) Internal carotid artery ligated.

Smear from right mastoid contains no microörganisms; culture, streptococcus non-hemolyticus. Smear from sinus, no microörganisms; culture, no growth.

*Operation followed* almost immediately by paralysis of left arm, leg, and lower portion of face.

Following day, incontinence. Very listless and hard to arouse.

Second day: Has very little fever. During day had slight signs of compression (pulse 66).

Third day: Death in deep coma, such as seen in diabetic toxemia, unrelieved by removal of catgut ligature from internal carotid or acute diabetic treatment. No signs of compression (*viz.*, slow pulse or ocular changes).

Autopsy refused.

*Comment:* The fatal outcome undoubtedly was precipitated by the arterial ligature, which abruptly interfered with a cerebral circulation already seriously crippled by arterial disease. In a similar case the proper procedure would be to occlude the artery temporarily by Crile's clamp, and its immediate removal upon the first symptom of vascular disturbance.

In the above case even the ligature of the internal jugular should have been omitted (the patient being a diabetic), as it was an unnecessary surgical trauma, especially as the thrombus of the bulb was aseptic.

(2) Thrombotic or embolic occlusion following dis-

lodgement of the clot which is formed just distal to the ligature.

The apoplectic seizure generally occurs from the third to the eighth day. It should be avoidable by carefully applying a ligature of fascia lata so as not to injure the intima of the vessel. (Dr. Ill's case.)

Case K.

*Hemiplegia from dislodgement of thrombus several days after ligation of common carotid.*

Patient of Dr. Edgar Ill.

Sloughing carcinomatous mass of ear and side of face involving dura; common carotid ligated to enable extensive curettement. On 8th day after operation, partial hemiplegia of opposite arm and leg; which almost completely disappeared in next few months, during which time radium entirely healed ulcer and relieved all pain. Death 18 months later from general carcinomatosis.

(3) Hemorrhagic from rupture of a cerebral vessel, the result of an aneurysmal dilatation following septic or toxic (mercurochrome) injury to the intima, thus weakening a cerebral blood vessel. (See Case M.)

This may occur at any time and is unavoidable as far as the sepsis is concerned, but warrants hesitancy in carotid ligature in all cases in which a foreign toxic material has been injected.

## OPERATIVE TECHNIC IN CAVERNOUS SINUS THROMBOPHLEBITIS

**Anterior Ophthalmic Cases.**—If the infection comes from in front (from a staphylococcic infection of the face, lip, or cheek), the orbit should be opened as widely as possible by a Kroenlein osteoplastic flap,

after ligation of the common carotid. To make multiple exploratory punctures of the orbital tissue is not only useless, but is apt to take away the chances of limitation of the infective process. The cardinal principle to remember is that *the infection is in the* vein. It should be inspected.

The post-mortem records show that in the majority of cases there is apt to be a small amount of pus at the apex of the orbit, and it is perfectly conceivable that if this is opened early, the sinus infection may be controlled in a small proportion of cases.

The writer believes he has thus cured two cases of cavernous sinus thrombosis by early operations, one by enucleating the eye, followed by serum therapy (Case 12), and the other by turning down a Kroenlein flap and evacuating a small amount of pus (Case B). In neither of these was a thrombosed vein seen.[1]

The orbital vein having been inspected and found thrombosed, or the decision reached that the sinus itself is affected by a purulent clot, all the orbital contents must be eviscerated; the sinus drained through the enlarged sphenoidal fissure by widening of this, as recommended by Mosher.[2] This is not a difficult operation, but it requires courage to remove an eye which is not yet blind.[3]

Case 22.

*Acute obliterating thrombophlebitis of cavernous sinus from acute ethmoiditis causing thrombophlebitis of ophthal-*

[1] Tervaert's case analyzed by Ritter.

[2] Mosher, Harris Peyton: "The Orbital Approach to the Cavernous Sinus," *Laryngoscope*, Vol. 24, 1914.

[3] Boenninghaus: *Loc. cit.*, pp. 230-231, recommends Moure or Preysing's operation for exposing the cavernous sinus.

*mic veins. Operation: Drainage of cavernous sinus from in front, associated with ligation of common carotid. Recovery.*

*Previous History:* Patient has always been subject to "colds in head, nose-bleeds, rose colds and hay fever."

*October 6:* Usual cold in head, which lasted three days.

*October 9:* Came home from school complaining of pain in nose and left eye. Temperature, 99° to 102° F. Treated for "neuralgia."

*October 10:* Left eye "began to swell."

*October 13:* Swelling of right eyelid increased. Pain in back of neck. Temperature 100° to 102° F. Ophthalmologist consulted.

*Diagnosis:* "Orbital cellulitis," as patient had slight exophthalmos and chemosis. Pain in back of neck.

*October 15:* Now complains of pain in shoulder and right groin, "swelling of left eye much less, but today slight swelling of right eye."

*October 16:* Dr. F. Chattin called. Diagnosis: Cavernous sinus thrombophlebitis because of "peculiar blueness of left lid which is different from that of orbital abscess." Admitted to infirmary.

*Examination: Face dusky.* Slight swelling of left side of face, but no tenderness. Cannot open jaw and holds head to left side because of soreness in neck and jaw; when moves arms, shoulder hurts her (pterygomaxillary infiltration).

Left lids swollen with dilated veins (the swelling of venous obstruction as well as inflammatory infiltration). Exophthalmos and chemosis. Movement of eyeball greatly limited. Fundus: white; margin of disc blurred; veins full. Vision: counts fingers at 3 feet. Right eye, slight edema of lids—with beginning exophthalmos and chemosis. Ocular movements limited, especially outward. Pupil dilated but reacts to light. Fundus, negative. (Few hours later, immediately before operation, marked papilledema with

dilated vessels.) Child also complains of leg feeling sore, but examination is negative. Temperature, 102.2° F. Pulse, 100. Leucocytes 25,000, polynuclears 84 per cent., lymphocytes 8 per cent. Blood culture, negative. Urine, marked trace of albumin.

*Operation:* Because of the difficulty in controlling hemorrhage at the apex of the orbit, while exposing the cavernous sinus through the orbit in Farr's case; and as ligation of the external carotid of the right side could not control bleeding from within the cranium, I ligated the common carotid as a preliminary procedure; during which the internal jugular was exposed and ligated, and the pterygomaxillary space explored—no pus found but enlarged glands and inflammatory infiltration.

*Orbital Operation:* Large Kroenlein osteoplastic flap of outer wall of orbit. Orbital contents eviscerated without hemorrhage. In upper part of orbit 15-20 drops of thick pus evacuated. Part of the great wing of sphenoid removed. Cavernous sinus exposed with anterior end of ophthalmic vein entering into it. Cavernous sinus opened and probe passed inward ¾ to 1 inch, until stopped by petrous pyramid. *No* bleeding; no clot.

Posterior ethmoid cells opened from orbit and found to contain pus. Ethmoid cells, sphenoid cells and orbit thrown into one cavity. Considerable nasal hemorrhage.

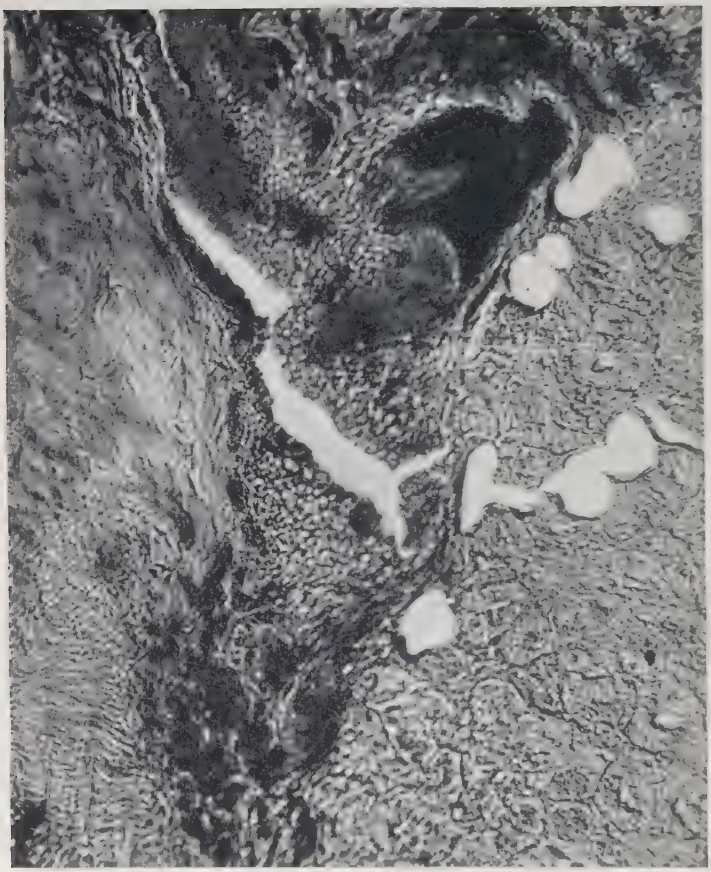
*Tissue Examination, Microscopic:* "Marked infiltration around attachment of ocular muscles, not around optic nerves."

*Microscopic:* Acute hemorrhagic inflammation of muscle bundles and other tissues. In one area of hemorrhagic infiltration, many of the cells were crowded with black pigment granules.

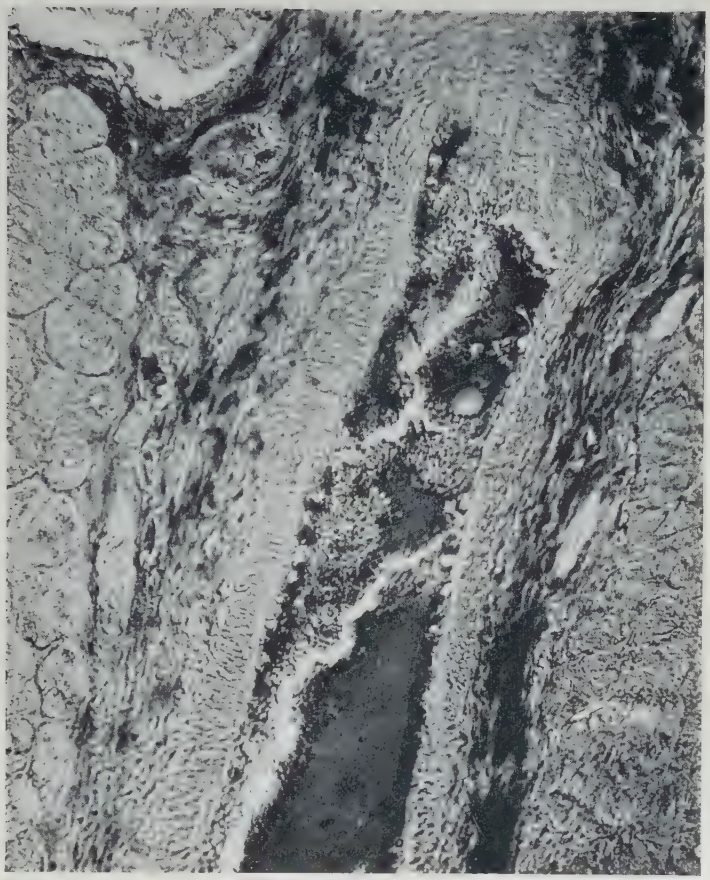
*Tissue Diagnosis:* "Thrombophlebitis of ophthalmic veins." Smear from cavernous sinus, a staphylococcus. (Dr. A. J. Eagleton.)

*Subsequent History:* The indistinctness of the disc margin and the fullness of the blood vessels of the funds of

Case 22.—Acute cavernous sinus thrombophlebitis



Case 22.—Thrombosed Orbital Vessels.



Case 22.—Thrombosed Orbital Veins.

the right eye persisted for several weeks. The blueness of the right side of the face, the exophthalmos and chemosis subsided almost immediately and disappeared within four days. The temperature reached normal on the third day.

**Sphenoidal Mesial Cases.**—When the infection comes from the sphenoid sinus, the cavernous sinus may be entered through the anterior wall of the sphenoid cavity, it being fully exposed by way of the lip (Cushing's trans-sphenoidal route)[4]; not through the small orifice of the nose.

To accomplish this the whole of the septum is removed and crushed against the turbinates, the sphenoidal sinus is opened and its mucous membrane removed, followed by removal of its posterior and lateral walls, and an incision teased into the cavernous sinus through this opening. To those who have operated on the pituitary this is not an extremely difficult proceeding. The only way to avoid fatal hemorrhage after exposing the dura is to feel one's way along millimeter by millimeter. To place a curette blindly into the sinus is suicidal.[5]

**Posterior (Aural) Cases.**—If the infection enters

[4] Cushing, H.: *Pituitary Body and Its Diseases,*" 1912, p. 297.

[5] Langworthy, H. G.: "Anatomic Relations of the Cavernous Sinus to Other Structures with Consideration of Various Pathological Processes by Which It May Be Involved," *Ann. Otol.-Rhin. Laryngol.,* Vol. 25, 1916, pp. 254-286. With complete bibliography. Suggests opening the sphenoidal sinus by means of a curette entering from the nose. Although his anatomical description is excellent, the topography of the sphenoidal sinus is so variable in its extent, both laterally and antero-posteriorly, that any such operation would, in my opinion, be unwarranted, and in a large proportion of cases it would not reach the sinus.

through the inferior petrosal, the jugular bulb having been widely opened, the sinus may be attacked from the floor of the middle fossa (Case 14),[6] by way of the inferior petrosal,[7] [8] [9] or after extenteration of the petrous pyramid if the labyrinth is affected and the facial nerve paralysed; but only after the whole region has been put at rest by ligation of the common carotid (Case 23).

Case 23.

*Chronic (compensatory) cavernous sinus thrombophlebitis of aural origin. Operation: evacuation of infection in petrosal sinus by opening jugular bulb. Placing parts at rest by ligation of common carotid. Recovery.*

*History:* Severe tonsillitis eight weeks ago accompanied by right otitis media with discharge; has been in bed off and on ever since. Two weeks later four chills, followed by irregular temperature (104° F. to normal), headache and discharging ear. (Typical history of chronic phlebitis of lateral sinus.)

[6] Dwight, Edwin Welles: "Thrombosis of the Cavernous Sinus," a report of four cases, including one cranial operation. *Boston Medical and Surgical Journal,* Vol. 146, 1902. Opened the sinus by the middle fossa following the route for extirpation of the Gasserian ganglion. The patient died within an hour.

[7] Ballance, Chas. A., and Hobhouse, Neil: A case of septic thrombosis of the left sigmoid, left cavernous, and left inferior petrosal sinuses, a suggestion for treatment in future cases. (Trans. Ninth Internat. Otol. Congress, Boston, 1912.) Opened the cavernous sinus in the middle fossa between the first and second branches of the fifth.

[8] Hartley successfully entered the sinus by the middle fossa in a case of non-infective thrombosis for tumor. Knapp and Hartley: "A Case of Traumatic (a doubtful one) Orbital Sarcoma, Followed by Aseptic Thrombosis of the Cavernous Sinus," *Archives of Ophthalmology,* Vol. 29, 1900.

[9] Richards, John D.: "Petrous Pyramid, Its Surgical Anatomy and Technic for Its Removal." Abstracted in *Laryngoscope,* November, 1924. Preliminary report: Tr. Amer. Laryng. Rhinol. Otol. Soc., 1907.

*October 11:* Chill few hours ago. Temperature, 102.2° F. Tenderness on mastoid; hearing reduced. O. E. vessels full and slight swelling of margin. No Kernig. Blood culture, streptococcus.

*Operation:* Perisinuous abscess. Thrombosed sinus—but no pus in sinus. Free bleeding obtained from behind —none from jugular bulb region. Excision of internal jugular.

*October 13:* Post-operative temperature, 99.4° to 101.3° F.; complains of some pain behind right eye. *Petechiæ* on arm.

*October 22:* Has been running irregular temperature, 98.4° to 103.3° F.—with one chilly feeling and one distinct chill. Has complained constantly of severe pain behind right eye, and slight headache.

O. E. right inner and upper margins of disc very indistinct. Veins very tortuous with flame-shaped hemorrhages along side of retinal vein. Left disc slightly indistinct in outer portion—veins full but no tortuosity. Right pupil slightly different from left.

No anesthesia over trifacial region. Slight Kernig.

Several blood cultures, negative.

| | | | | | |
|---|---|---|---|---|---|
| | | Polynu- | | Lympho- | |
| Leucocytes, | 6400 | clears, | 60 per cent. | cytes, | 34 per cent. |
| " | 5500 | " | 65 per cent. | " | 33 per cent. |
| " | 7400 | " | 60 per cent. | " | 36 per cent. |

*Diagnosis:* Chronic compensating superior petrosal thrombophlebitis with cavernous sinus involvement.

*Operation:* Ligation of common carotid to place part at rest and control hemorrhage. Exposure of jugular bulb, from which obtained free pus—no bleeding from inferior petrosal.

*Subsequent History:* Temperature went to normal almost immediately. The pain behind the eye continued off and on, but was greatly modified for three weeks. The fundus cleared in about the same time.

*Surgical treatment of thrombophlebitis of large cerebral sinuses in which the blood current is not completely stopped.*

*Operative Technic in Mural Clot:* In parietal or mural infected clot the surgeon must first control the adjacent infection by the elimination of the bony caries, and then remove, if possible, the infection within the vein by small incisions through it at the site of the original point of infection. In my experience, the infection in the majority of cases can thus be limited. The slight bleeding which follows perforation through the clot is easily controlled by the placing of a small plug of cotton over the aperture, thus allowing the reëstablishment of the circulation.

If, however, the circulation in the sinus is obliterated by firm pressure (as must be applied if a large incision is made into the sinus), the stoppage of the blood current is apt to cause an extension of the infection in the sinus wall, as the protective mechanism of the circulating blood has been removed by pressure.

On the other hand, if a small incision is "teased" in the sinus (so that any thickening of the sinus wall or mural clot may be discovered) on the appearance of the jet of blood which shows that the sinus is still pervious, the opening is covered by a plug of cotton—on removing the cotton a few minutes later the bleeding will not recur, the small incision having been closed by the sliding of one wall of the sinus over the other.

## OPERATIVE TREATMENT OF CAROTID VENOUS PLEXUS CASES THROUGH THE FLOOR OF THE MIDDLE FOSSA.

**Technic of Opening the Cavernous Sinus Through the Floor of the Middle Fossa.**—(a) Usual Gasserian ganglion incision. (b) Location of the 3rd branch of the trifacial extradurally,[10] and of the crotch

[10] Frazier, Charles H.: *Surgery of Trigeminal. Keen's System of Surgery,* Vol. VIII, p. 190.

between the 2nd and 3rd branches. (c) Thorough exposure of the 2nd branch as it enters the foramen rotundum. The anterior portion of the Gasserian ganglion is now directly external to the sinus, while mesially the bony floor rises rather abruptly toward the sella. (d) If the dura is now incised directly under the ganglion by a semi-blunt knife, cutting on the flat at right angles, the cavernous sinus is opened in front and below the carotid artery as it emerges posteriorly from the carotid canal below, and then bends shortly as it takes its horizontal course forward in the sinus.

### INFECTION IN THE PTERYGOMAXILLARY FOSSA AND ITS RELATION TO SURGERY OF THE CAVERNOUS SINUS, ESPECIALLY IN CASES OF CAVERNOUS SINUS THROMBOPHLEBITIS ORIGINATING FROM THE INTERNAL CAROTID VENOUS PLEXUS.

The pharyngomaxillary space is an irregular cone-like region of considerable size filled with cellular tissue bounded by reflection of the deep fascial layers; the base of the cone being limited upward by the inferior surface of the petrous pyramid. The cone gradually tapers downward to a point where the fascia encircles the deep vessels, the internal jugular and the carotids, at about the level of the angle of the jaw.

The stylopharyngeus and the styloglossus muscles divide the pharyngomaxillary space into a large anterior compartment, which contains the tonsil, and a smaller posterior one containing the deep vessels. (Zuckerkandl.)

The external surgical boundary of the pharyngomaxillary space is made up of a reflection of the deep

fascia: it has great resistance. In its upper portion the space is partially covered by the parotid gland and is crossed by the facial nerve. Its internal boundary is formed by the superior constrictor of the pharynx.

A firm layer of prevertebral fascia covering the vertebral muscles separates the pharyngomaxillary from the retropharyngeal space; the latter being situated mesially to it on the posterior wall of the nasopharynx.

The retropharyngeal space, in its turn, is separated from the mucous membrane of the pharynx by a continuation of the prevertebral fascia covering the recti and longus colli muscles of the vertebræ.

At the level of the hard palate the retropharyngeal space is more prominent, but at the level of the tongue the pharyngomaxillary space is larger.[11]

The prevertebral fascia is so firm that suppuration within the retropharyngeal always travels downward and never perforates into the pharyngomaxillary compartment.

The retropharyngeal space contains a lymph gland, which continues during life, all the other numerous glands present in infancy in this region disappearing during the first year. True retropharyngeal abscess of glandular origin is consequently a disease of infancy.

There are, however, lymphatic nodes in the pharyngomaxillary space situated internal and anterior to the carotid, between the latter and the prevertebral fascia.[12] [13]

[11] Eycleshymer, A. C., and Schoemaker, D. M.: *Cross-section Anatomy.* New York, Appleton & Co.

[12] Woolsey, George: *Applied Surgical Anatomy,* 1902, p. 111.

[13] Moure, E. J.: *Diseases of the Pharynx and Larynx,* 1909, p. 131. Escot's Diagrams of Retropharyngeal Space.

**Upper Portion of Pharyngomaxillary Space.**—The base of the skull at the point of exit of the jugular bulb and internal carotid artery is over one-half inch above the cranial articulation of the atlas.

It is this upper portion of the pharyngomaxillary space which is of surgical importance in thrombophlebitis of the cavernous sinus following infection from the jugular bulb, the internal carotid venous plexus, or the inferior petrosal.

Exposure by dissection of the facial nerve as it loops forward from the stylomastoid foramen into the parotid gland, followed by the introduction of a Killian spatula above the loop, will reveal the large extent of the upper portion of the space.

The upper portion of the pterygomaxillary fossa contains the internal jugular, mesial to which is the internal carotid artery. The surgical guide is the styloid process, the vessels being anterior to it.

Mesially to the point of exit of the internal carotid and opposite the bony exit of the Eustachian tube, the space is entered by the pterygoid plexus, communicating with the cavernous sinus through the foramen spinosum; the veins that perforate the foramen lacerum medium being still more mesial, while the pharyngeal plexus is further forward.

Suppuration in the pharyngomaxillary space after the first year is apt to be of hemic circulatory origin—either from a phlebitis of the jugular bulb of the veins emptying into the internal jugular, which has tributaries to the walls of the pharynx from the venous plexus of the internal carotid, or one of the pharyngeal or pterygoid plexus from thrombo-venous phle-

bitis of the veins of the foramen ovale or veins of Vesalius.

Phlebitis of the tonsillar veins is situated much lower in the space and generally external to it.

The size of the space enables it to be the site of suppuration without being visible on oral inspection because of its high position, or externally without fluctuation because of its great depth. When, however, the pus crowds the inner boundary of the space into the nasopharynx, the abscess may be manifested by a nasal voice, irregular difficulty in swallowing, and a swelling in the lateral posterior wall of the naso-pharynx above the line of the palate.

An abscess of the pterygomaxillary fossa can be distinguished from a true retropharyngeal abscess by the former being situated in the lateral and posterior wall of the naso-pharynx, above and posterior to the tonsil; the latter, while being partially localized to one or the other side of the median line, is situated lower down, below the palate. A sphenoidal sinus mucocele that perforates the anterior wall of the sinus is situated very near the median line but high up behind the palate.

The pterygomaxillary fossa can be opened from below along the anterior border of the sternocleidomastoid.[14] This should be performed under local anesthesia, as experience shows that a large number of these cases die suddenly during operation; death occurring not from respiratory obstruction alone but from sudden cessation of both respiration and heart.

[14] Dean, L. W.: "The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebræ," *Annals of Otology, Rhinol. and Laryng.*, June, 1919, p. 566.

CASE L.

*Sudden death following opening of the pharyngomaxillary fossa.*

Seen in consultation with Dr. Henry Barkhorn and Dr. C. A. Wallenstein.

*History:* Had what was thought to be a quinsy sore throat; opened anteriorly obtaining pus. A few days later again opened posterior to tonsil. Following operation septic elevations of temperature and sweats. Blood culture, negative. Later had what "looked like a bite on the side of his face." This gradually developed into a large abscess under the temporal muscle; opened under local anesthesia, evacuating a large quantity of pus from temporal fossa. Second blood culture, streptococcus hæmolyticus. Recently has had two chilly sensations followed by marked elevation of temperature. Has no pain excepting in the side of his face where the swelling had occurred.

*Examination:* In looking into his mouth an enormous swelling of tonsils; pus is coming from in front and behind it! There is also a large amount of pus coming out from under the temporal muscle whenever he opens his mouth. "He must have an involvement of his pharyngomaxillary fossa with a jugular vein thrombophlebitis."

*Operation:* (H. B.) Ether. Followed jugular vein to base of skull. Phlebitis of jugular exposed; just as operator entered the pharyngomaxillary space (which was boggy but contained no pus), patient stopped breathing and heart stopped.

ANATOMICAL ILLUSTRATIONS OF PTERYGOMAXILLARY FOSSA

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Case 24.

*Cavernous sinus with pterygomaxillary fossa abscess infection apparently extending by carotid venous plexus.*

Patient of Dr. E. A. Curtis.

*Previous History:* Frequent sore throat. No previous ear trouble.

*History of Illness:* Wednesday, Sept. 23, earache with slight discharge and some deafness. Following night child slightly delirious; and on third night very delirious; Saturday, Sept. 26, paracentesis; followed by discharge of a large amount of fluid, the subsidence of the temperature

and the disappearance of all pain; but slight redness and puffiness developed above the mastoid.

*September 30* (7th day): Slight chill during day, and severe chill at night.

*October 1:* Temperature normal. *Urine:* Trace of albumin.

*First Operation:* Simple mastoid (Dr. E. A. Curtis). Very little pathological change, with the exception of a small amount of granular tissue over the lower portion of the sigmoid sinus.

Following operation, repeated sudden rises of temperature to 104° or 105° F., followed by remissions, without sweating but associated with very rapid pulse, 130-150.

*October 2* (9th day from initial earache and 2nd day after operation): Irritable and restless with intervals of drowsiness. Slight Kernig; and stiffness of neck. *Slight exophthalmos,* with suggestion of weakness of left lower face. (Dr. J. V. Bissett.)

*Blood:* Leukocytes 15,000, polynuclears 90 per cent., lymphocytes 10 per cent. Culture, negative.

*October 3: Exophthalmos has disappeared,* but child is restless. Intermittent septic temperature and very rapid pulse continue.

*Second Operation:* (Drs. Curtis and Brien.) Jugular vein ligated, lateral sinus explored; no clot.

*October 5:* Vomiting. *Examination:* Continued weakness of left lower face. Fundus: margins of both discs slightly indistinct, especially on left side. *Blood:* Leucocytes 16,000, polynuclears 89.5 per cent., lymphocytes 10 per cent., large mononuclears 5 per cent. Second culture, negative. Blood transfusion, 200 c.c. from immunized donor.

*October 7: Recurrence of left-sided exophthalmos;* slight ptosis, and swelling of the left upper lid, which is tender on pressure. Papilledema, especially of left, with dilated veins.

*October 9:* Left side of face is now swollen, and the edema of left lid has greatly increased. The left eyeball

is on a different level than the right eye. The child apparently cannot move his left eye well outward. The veins of left disc are larger than previously.

Patient continues slightly stupid and irrational, with suggestion of Kernig and stiff neck.

*October 10:* Ptosis is undoubtedly better. Temperature, however, again reached 105° F., and pulse continues disproportionately rapid.

*Preoperative Diagnosis:* "Cavernous sinus thrombophlebitis and protective meningitis; following inferior petrosal sinus involvement secondary to a primary jugular bulb phlebitis; the infection having entered the venous system through a dehiscence in the floor of the middle ear on September 30 (the seventh day of the disease). (The diagnosis of floor dehiscence rests on the patient being a child, the rapid onset of meningeal symptoms, and the negative operative lateral sinus punctures.)"

*Comment on Diagnosis:* The diagnosis should have been *cavernous sinus obstruction, the result of primary jugular bulb thrombophlebitis with extension through the carotid venous plexus.* This diagnosis was called for by the early development and transient character of the exophthalmos; as, had the infection from the bulb travelled through the inferior petrosal (as was supposed), it would have attacked the *posterior part of the cavernous sinuses* and not given rise to an exophthalmos; but an extension through the carotid plexus (as was the case) primarily reaches the anterior portion of the cavernous at its junction with the ophthalmic, giving rise to early and transient exophthalmos.

*Third Operation:* (W. P. E.) Preliminary ligature of the common carotid, just below the bifurcation. The neck operation revealed a thrombosed vein just anterior and external to the carotid artery, which was doubly ligated.

*Cranial:* Incision through the lower portion of the sigmoid sinus evacuated considerable pus; no bleeding obtained from below. Culture from mastoid, non-hemolytic streptococcus.

Operation was followed by blood transfusion (second).

*Following day, ptosis, exophthalmos and swelling of lid have entirely disappeared, and eyeball movements are apparently normal;* but child is extremely restless, somewhat delirious, has vomited several times, and has involuntary micturition.

*October 15:* Child complains that "neck hurts him." He has a general septic appearance; the remittent septic temperature has continued; and he has meningeal symptoms —rapid pulse, involuntary defecation—although he has never complained of pain in head. Third blood cultures, negative.

*Second Bulbar Exploration:* The reopening of the neck wound liberated a quantity of pus, from high up toward the stylomaxillary fossa, which was thought to come from the upper end of the ligated jugular vein itself, although this could not be demonstrated. Palpation with finger well up to base of skull revealed nothing. Exploration of the jugular bulb region, during which an opening into the dura of the posterior fossa was accidentally made, liberating a large quantity of cerebrospinal fluid. Unsuccessful attempt to irrigate water from the opening in the sinus above through the jugular bulb into the neck wound (in order to eliminate an accumulation of pus in the jugular bulb).

*October 16:* Since last exploration complains of pain in throat, is not able to swallow fluids, and coughs frequently. "May be due to trauma from finger in stylomaxillary space."

*October 17:* Glandular swelling on both sides of neck. Left side slightly more prominent than right.

*October 19:* Nasopharyngeal palpation reveals soft mass in the lateral and posterior wall of the nasopharynx. "It may be due to a secondary abscess from the cavernous sinus phlebitis involving the nasopharynx, or an inflammatory swelling from external neck wound."

*October 21:* Yesterday had a chill, and today another, the first since the period of primary invasion (September 30 and October 1), both followed by sudden ascent of temperature from 98.2° to 106.2° F.

Sixth blood transfusion of immunized blood (200 c.c.).

Examination in sitting position reveals paralysis of left trapezius (the scapular edge being too far out).

*Note:* Largely from the trapezius paralysis a diagnosis was made of "a septic focus existing in the jugular foramen, which occasions pyemia and is associated with an inflammatory exudate causing pressure on the vagus and glossopharyngeal, thus giving rise to the rapid pulse and paralysis of the throat."

*Third Ether Exploration:* Wiping the extensive suppurating neck wound was followed by a tremendous hemorrhage from the carotid artery, which had become divided by sepsis at the site of the ligation. The hemorrhage exsanguinated the child. Its control necessitated the ligating of the carotid below the original wound and the application of a ligature on the external carotid above.

Operation immediately followed by a transfusion of 200 c.c. (seventh).

*Comment:* Following the accident, as the child did not complain of any pain in the throat, as the difficulty in swallowing had largely disappeared, and repeated investigation (Dr. Curtis) in the upper end of the neck wound failed to reveal any external swelling or pus; no further attention was paid to the small soft mass in the upper and lateral wall of the nasopharynx, which had been felt on October 19; the irregular dysphagia being accounted for on the supposition of a glossopharyngeal paralysis from pressure on the nerves passing through the jugular foramen.

*October 24:* Chill (fifth). Delirious at times.

*October 31:* Chill (sixth); vomited. Great restlessness.

*November 1:* Fluid again returning through nose. Severe choking spells. Delirious; neck stiff. Optic discs choked, with large veins more marked than at last examination. Ninth transfusion.

*November 2:* "Terminal meningitis." (Meningeal cry. Retraction of head. Tendency to lateral deviation to left and upward. Vertical nystagmus on looking to right.) "Voice has the rough sound like palatine paralysis."

Lumbar puncture: Cloudy cerebrospinal fluid under great pressure, coagulable albumins + + + +, globulin increased + + +, chloride .61 mg., contains 1,430 cells, chiefly polynuclears, streptococci + + +. Culture, streptococcus non-hemolyticus.

*November 3:* Death.

*Post-mortem: Venous Sinuses:* Jugular bulb, petrosals, and the left lateral sinus (the infected side) are free from pus. The cavity of the jugular bulb is not to be found. Splitting open the left inferior petrosal shows that its outer end is obliterated and free from pus, but the inner extremity where it enters the cavernous sinus, contains a slight amount of pus. The left superior petrosal is free; its lumen is not obliterated.

*Cavernous Sinuses:* The left cavernous sinus is distended; its incision liberates a quantity of pus. Pus in sinus confluence which reaches to the right cavernous sinus, the wall being thickened and necrotic; and its cavity distended with pus.

*Opposite Petrosals:* The right superior petrosal is obliterated by exudate; no pus.

*Base of Skull:* The left carotid canal contains pus. The nerves and vessels that pass through the foramen lacerum medium on the left side are surrounded by pus. The ganglion of the 5th nerve of the left side is edematous.

*Sphenoid:* The bone over the sphenoid is grayish; opening the sphenoid cavity reveals free pus [15] (grumous). The apical cells of the left petrous are free.

*Nasopharynx:* Opening the nasopharynx from the cranial cavity above reveals a large abscess in its lateral wall, which contains an ounce of pus, and *apparently communicates with the carotid canal.*

*Brain and Coverings:* Meningeal exudate chiefly over the left cortex, running from the temporosphenoidal lobe upwards. Meninges of right hemisphere edematous.

*Basal Cisterna:* Excessive cerebrospinal fluid. The supe-

[15] See Note 6, O'Malley, on p. 13.

rior margin of the cerebellum is covered with exudate. The right cerebellum at the inferior external surface has a little exudate. Cisterna magna is free. (In a general way, the process is limited to the cortex of the hemisphere of the affected side and to the external and inferior surface of the cerebellum of that side, although it extends to the opposite side.)

### SYNOPSIS OF EXTENSION OF INFECTIVE PROCESS IN VIEW OF HISTORY AND POST-MORTEM FINDINGS

*September 23:* Middle ear infection followed by a primary jugular bulb thrombophlebitis associated with a protective meningitis of the basal cisterna (as shown by the symptoms of meningeal irritation, restlessness, drowsiness and rapidity of pulse during the first week).

The infection rapidly spread through the carotid plexus. (Its virulence was evinced by the rapidity of the pulse, which was out of proportion to the temperature from the beginning of the illness, by the extension of the infection through the carotid canal, by the post-mortem findings, the petrosals not being involved.)

*October 2:* Nine days after the initial earache the infective process must have been present in the anterior walls of the cavernous sinus sufficient to cause obstruction in the return circulation from the ophthalmic and pterygoids (as manifested by slight left exophthalmos and facial weakness).

The venous current was probably almost immediately reëstablished by collateral circulation (as the exophthalmos disappeared on the following day). The suggestion of left lower facial weakness was possibly due to a primary venous obstruction of the pterygoid plexus.

A septic process in the cavernous sinus itself was certainly present by October 5. The cavernous sinus was definitely obliterated by *October 7* and probably at this time contained pus.

Suppuration in the pharyngomaxillary space was undoubt-

edly present on October 15 and may have been earlier; (it resulted either (1) from a retrograde thrombophlebitis of the pterygoids by way of the accessory veins through the foramen ovale, foramen lacerum and Vesalius' veins, (2) from nutritional death following the ligation of the carotid, or (3) from an associated sphenoidal sinus suppuration).

The separate processes probably were more or less interdependent one on the other; although the position of the abscess in the lateral wall rules out the sphenoid sinus as its direct cause.

*Comment:* Of chief importance is the relationship of the retropharyngeal abscess and its possible localizing value as to the position of the septic causative focus. Properly interpreted, it should have definitely localized the suppurative focus in the middle fossa, toward the median line, to which all surgery should have been directed after the exploration of October 10.

A correct diagnosis having been made, the proper surgical treatment would have been (1) drainage of the retropharyngeal abscess by external route on October 15.

This failing to release the sepsis, (2) the cavernous sinus itself should have been opened either by sacrificing the eye or through the floor of the middle fossa.

As the surgeon will of necessity shrink from sacrificing a normal eye in an aural case; an attempt to drain the cavernous sinus from the floor of the middle fossa should have been made.

# CHAPTER XIV

## TREATMENT OF THROMBOPHLEBITIS BY SEROTHERAPY AND VACCINOTHERAPY

### TREATMENT BY MERCUROCHROME-220, GENTIAN VIOLET, ACRIFLAVIN

The nature of the pathological anatomy of thrombophlebitis of the basilar sinus would appear to be favorable for treatment by sterilization of the blood stream by the intravenous injection of bactericides, because the disease is within the blood vessels themselves.

Experimentally it has been demonstrated that in mice about 25 per cent. of the cases of induced blood stream septicemia from streptococcus hemolyticus, which otherwise are uniformly fatal, are saved by injection of mercurochrome. (Kolmer.[1]) Clinically there is evidence that mercurochrome (although far from a universal specific) exerts a favorable influence on a certain proportion of cases of general blood stream infection.[2]

This, combined with the demonstrated efficiency of chemotherapy in eradicating the trypanosomes of syphilis and yaws from the circulation, has stimulated

[1] Kolmer, John A.: "The Chemo-therapy of Bacterial Diseases." Address before the Essex County Anatomical and Pathological Society, New Jersey, April 23, 1925.

[2] Halphen, E.: Thrombophlébite du sinus caverneaux consecutive à une otorrhoé chronique réchauffeé. Injected trypaflavin intravenously, with slight improvement. *Arch. Intern. de Laryngol.*, Paris, 1924, Vol. XXX, pp. 167-172.

a large number of men to experiment in both clinic and laboratory; and warrants the prediction that, in the near future, there will be discovered specific chemical compounds which will influence the different pyogenic infections of the blood stream, either by inhibiting their growth or endowing the blood with greater bactericidal activity.

However, I think it is a fair inference to predict that the "therapia sterilisans magna" when discovered will be specific only for certain types of the usual micro-organisms causing blood stream infection, as quinine is for malaria, and 606 for syphilis. To-day none of the known chemo-therapeutic agents—mercurochrome, gentian violet, acriflavin—penetrate into experimentally produced edematous tissue in sufficient amount to be bactericidal (Hirschfelder, Malmgren, Creavy);[3] and clinically they have all failed when the site of the infection is in the coats of the veins—periphlebitis, or is a localized focus outside of the circulation—perisinuous abscess, or even in freely moving blood when attacked by certain micro-organisms—viridans and pneumococcus.

Case 25.

*Blood stream infection of 57 days' duration from right-sided pneumococcus endocarditis; erroneously diagnosed as cavernous sinus thrombophlebitis, without exophthalmos secondary to spheno-ethmoidal disease (because the onset was seemingly from a nasal suppuration). Apparently temporary sterilization of blood stream by intravenous injection of mercuro-*

[3] Hirschfelder, Malmgren, Creavy: "Penetration of Mercurochrome, etc., into Edematous Tissue," *Journ. Pharm. and Exper Therapy,* Vol. 24, p. 457, 1925.

*chrome; the post-mortem showing a healed thrombosis of the cavernous sinus, which may have been the portal of entry. Post-mortem.*

Synopsis of report by Dr. Royal A. Schaaf.

*Synopsis of History:* For about three months previous the patient had "nasal catarrh," which during the week immediately preceding the last illness became markedly worse; accompanied by profuse nasal discharge and severe frontal headache. Sudden chill, followed by marked rise in temperature and profuse perspiration.

On the fifth day, had a second chill, with fever, both of which recurred repeatedly until death, on the fifty-seventh day of the disease.

*Course:* Ague-like paroxysms recurring at irregular intervals, at times several in twenty-four hours. Sudden rise in temperature from normal, the peak being reached within an hour after the chill. Temperature, 103° to 106° F., in different seizures. Followed by profuse sweats.

*Embolism:* During the first three weeks of the illness, evidence of embolism; *viz.*, arthritis of the shoulder, metatarsophalangeal and temporomaxillary joints. All embolic foci cleared up in a few days after their appearance without showing any tendency to suppurate. Petechiæ were never present. After the third week, except for the occasional presence of a few moist rales in various areas of the lungs, following a chill, there was no further suggestion of embolism.

*General Condition:* Between chills his appearance belied the actual condition.

All cardiac, pulmonary, gastro-intestinal and mental symptoms were conspicuous by their absence. No signs of meningeal irritation were observed.

*Examination: Eyes:* Eye ground showed bilateral papillitis. *Nose:* Some discharge, particularly from the left nostril. X-ray revealed cloudiness of the left frontal and ethmoid sinuses and of both antra. *Lungs:* A few fleeting moist rales in various areas followed some of the febrile paroxysms until the terminal pneumonia in the right lower

lobe on the fifty-fifth day. X-rays of the chest (December 12) negative. *Heart:* Normal until last three weeks of the illness. On thirty-seventh day of disease a faint double murmur over the pulmonic area in the second left intercostal space, closely resembling the friction murmur of a dry pericarditis. The murmur increased in intensity for several days and then remained unchanged until death. *Abdomen:* Normal. *Spleen:* never palpable.

*Laboratory Findings: Urine:* Early in the illness, gross blood, attributed to an infarct in the kidneys. Subsequently, many analyses showed one or two plus albumin, a few leucocytes, an occasional hyaline or granular cast. *Blood:* Marked leucocytosis: total count, 16,000 to 42,000; differential, 88 per cent. to 94 per cent. polymorphonuclear neutrophiles. Blood culture: First (December 4), showed no growth after five days. Second and third (December 10 and 12) swarmed with greenish, non-hemolytic colonies on blood glucose plates in 24 hours. Smear showed large cocci and diplococci in chains; no capsule; bile soluble. Precipitin tests with Group I, II, and III pneumococcus serum, negative. No growth in subcultures in plain broth. Serological diagnosis: Pneumococcus. Group IV.

*Chemo-therapy:* Intravenous administration of three doses of 350 mg. of mercurochrome (December 13, 15, and 24). The blood culture taken the day following the second dose was negative, although the culture before the first dose showed a tremendous number of colonies. At the time it was thought that mercurochrome had sterilized the blood stream, but later events proved that at most it may have aided in the sterilization of the peripheral blood stream only, it being without effect on the septic focus. Each time the drug produced vomiting and profuse watery diarrhea lasting twenty-four hours. There was also a chill in each instance, but it could not be determined whether this was due to the disease or to the mercurochrome. There was only a slight and brief change in the character of the temperature curve after the injections.

Fifth blood culture (December 19), positive, an average

of eight colonies per cc. of blood. 6th and 7th cultures (December 22 and 29), negative. Transfusion of 750 cc. of whole blood (January 3). 8th culture (January 10) showed an average of 130 organisms per cc. of blood. (Clinical symptoms apparently bore no relation whatever to the condition of the peripheral blood as shown by culture.)

*Diagnosis:* (W. P. Eagleton.) "Septic thrombophlebitis with a mural clot, involving one or both cavernous sinuses, from spheno-ethmoidal disease, without sudden venous obstruction," the diagnosis being made because the sepsis apparently dated from the nasal suppuration and was accompanied by bilateral papilledema. It was thought that malignant endocarditis was eliminated by the absence of petechiæ, enlargement of the spleen or evidence of involvement of the heart.

*Autopsy: Brain meninges and venous sinuses,* normal except for *a small organized apparently non-septic clot on the wall of the left cavernous sinus.* Left ethmoid and frontal sinuses moist, but no accumulation of pus. *Heart:* On the pulmonary valve, a single huge vegetation, *the sole cause of the septicemia.* Cultures taken from the vegetation yielded an abundant growth of an organism identical, in all respects, with that found in the blood cultures during life. Pericardium and left chamber and valves, normal. *Lungs:* Lobar pneumonia involving the right lower lobe accompanied by pleurisy with effusion. Numerous embolic areas diffusely disseminated throughout both lungs, none of which shows any tendency to suppurate.

*Comments on Post-mortem:* It is possible that the cavernous sinus thrombosis was embolic. But as the history of invasion was undoubtedly of nasal and then of cerebral origin, and as a septic vegetation limited to the right heart alone is almost unknown (although fairly common secondary to a left-sided valvular infection), the inference is forced that the entrance of the pneumococcic infection into the blood stream was by way of the basal sinus; especially

as general pneumococcic sepsis (in the absence of pneumonia) is frequently of nasal accessory sinus origin.[4] [5]

It is probable that after invasion of the left cavernous sinus colonies of micro-organisms descending through the internal jugular found a site for growth on the right valve. The limitation of the vegetative process to the right heart undoubtedly explains the absence of petechiæ or splenic enlargement, and the great variation in the blood culture findings, as the bulk of the bacteria and emboli were evidently filtered from the blood in its passage from the right heart through the lung, the organism thus being rapidly destroyed in the blood stream.

The first intravenous injection of mercurochrome apparently sterilized the blood stream, but it certainly had no influence on the focus on the right heart valve, although it may have been the curative agent within the cavernous sinus itself; as the slowness of the circulation within the sinus may have given time for the action of the drug, which was not present during the rapid passage of the blood stream through the heart.

## DANGERS OF INJECTION OF TOXIC FOREIGN CHEMICAL AGENTS IN SEPTIC INVOLVEMENT OF THE BLOOD VESSEL OF THE BRAIN

The treatment of thrombophlebitis of the basal sinuses by a toxic chemical agent must, of necessity, be more hazardous than when the infection is in the less specialized parts of the body. For the blood vessels of the brain not only supply its nourishment but on them depends its structural integrity. They are the framework of the brain, from which is suspended, as it were, the nervous tissue. Consequently, any dis-

[4] Darling, S. T.: "Abscess of Nasal Sinuses and Pneumococcic Infection," *Jour. A. M. A.*, Vol. 47, p. 1561, November 10, 1906.

[5] Felty and Keefer: "Pneumococcus Sepsis in Absence of Pneumonia, *Jour. A. M. A.*, Vol. 81, 1601, November 10, 1923.

turbance of the circulatory system, already overtaxed by the disease, may be associated with most disastrous effects.

The anastomosis of the arterial supply to the brain is so complete that surgical obliteration of any one set of large vessels will generally not seriously interfere with its function [6] (so long as the intima is not affected); this also applies in a much greater degree to the venous return, although occasionally upset disastrously by surgical intervention.[7]

However, profound blood stream toxemia, as in certain cases of thrombophlebitis, has a destructive action on the endothelium of the vessels of the pia, as shown by the multiple hemorrhages into the cortex in cases of fulminating meningitis;[8] and mercurochrome, in common with all mercurial preparations, weakens by its toxic action the intima of the delicate vessels of the brain, also giving rise to multiple small hemorrhages (demonstrated in the lung by intratracheal injections).[9]

Thus, either or both of these conditions may be the underlying factor in causing rupture of a large cerebral artery in the presence of a neighborhood focus of infections—sinus thrombophlebitis or brain abscess—

[6] Fay, Temple: "The Cerebral Vasculature" (Preliminary Report of Study of Means of Roentgen Ray), *Jour. A. M. A.*, Vol. 84, No. 23, pp. 1727-1730, June 6, 1925.

[7] Eagleton, W. P.: "Circulatory Disturbances Following Ligation of the Internal Jugular Vein in Sinus Thrombosis with Report of a Case," *Arch. of Otology*, Vol. 35, No. 2, 1906.

[8] Eagleton, W. P.: "Meningitis," *Ann. Oto. Rhin. and Laryn.*, 1917, Vol. 26, p. 1060. Tr. New York Otological Soc., January, 1917.

[9] Cooper, H. J.: "Pathologic Changes in Lung from Use of Mercurochrome-220," *Jour. A. M. A.*, Vol. 85, p. 256, July 25, 1925.

especially if the blood supply is further upset by surgical interference from ligation of the carotid.

This occurred in the following case which demonstrates the special dangers of disturbance of the circulation in the presence of infections within the dura.

CASE M.

*Pyemic sepsis and localized meningitis from labyrinthitis; treated by intravenous injection of mercurochrome (with severe reaction). Ligation of the right common carotid and the internal jugular vein, associated with exploratory punctures of the posterior and middle fossa; followed three days later by right hemiplegia from hemorrhage into ventricle, secondary to a small area of cerebral infective softening; the contributing causes probably being the mercurochrome and the ligation of the common carotid. Autopsy.*

Patient of Drs. A. Stahl and William Brien.

*Previous History:* Four years previously the patient had bilateral otitis media, associated with a left facial paralysis; which latter disappeared, leaving, however, a bilateral chronic suppuration with deafness of the left ear.

*Present Illness* of one month's duration with acute beginning exacerbation of right otitis accompanied by severe pain in head and profound deafness (as she depended on the right ear since the previous attack). The pain in head was so severe at times that the patient became maniacal, thus masking an associated dizziness and nausea, so that little attention was paid to the latter symptoms. A few days prior to admission, the right side of her face suddenly became paralysed.

*January 2, 1925:* Admitted to Eye and Ear Infirmary with spontaneous nystagmus; right facial paralysis. Hearing: right, negative. Left, reduced to ability to hear a loud voice.

*Cold Caloric:* (Dr. Brien.) Right apparently does not

alter the spontaneous nystagmus. Left apparently has slight effect on the left vertical and horizontal canals.

*Diagnosis:* Acute exacerbation of chronic otitis with labyrinthitis.

*Operation:* (Drs. Brien and Stahl.) Radical mastoid, right, granulations and cholesteatoma in middle ear and mastoid; sinus and dura of middle fossa thickened and covered with pus and granulations.

*January 3-6:* Stiff neck; pain in head, restlessness. Temperature, 100° to 102° F. Pulse, 86-110.

*January 7:* Slight chill; vomited. Temperature following chill reached 102.2° F. Blood count: leukocytes 6,600. Polynuclears 80 per cent. Small lymphocytes, large lymphocytes and large mononuclears, 20 per cent. First blood culture, negative.

*January 8:* Second chill. Temperature, 101° to 102.2° F., the latter being the highest recorded. Treatment: intravenous injection of mercurochrome, 10 cc. of 1 per cent. solution administered at 12 o'clock noon. Followed by severe reaction—chill, and at 2:30 p. m. temperature, 105.3° F.; pulse, 120; 4 p. m. temperature, 106.2° F.; pulse 134; 5:00 p. m. temperature, 105.2° F.; pulse, 130. Blood pressure, 98/70; with frequent urination and mercurochrome in specimen in 2 hours. (See autopsy report.) Lumbar puncture: pressure increased, fluid turbid. Coagulable protein present, globulin + + + +. Cells per c.mm. 5,920. Polynuclears 80 per cent; lymphocytes, 20 per cent.; sugar, normal; globulin increased + +. Smear, diplococci; culture, feeble growth of staphylococcus albus.

*Diagnosis:* "Labyrinthitis and suppurative meningitis, secondary to brain abscess, probably of the posterior fossa."

*Operation:* (W. P. E.) Ligation of right jugular vein and common carotid artery. Removal of solid angle and entire roof of the tegmen, so that a large area of the under surface of the temporo-sphenoidal lobe was exposed. Considerable bleeding from accidental opening of the lateral and superior petrosal sinuses; controlled with difficulty.

Fistula in the horizontal semicircular canal; the semicircular canals looking dark, probably from destructive labyrinthitis. The whole brain was very tense and did not pulsate; but after a lumbar puncture it pulsated in a normal manner. Elevation of the dura disclosed a granulating extradural mass directly above semicircular canal. Through this an opening into the subdural space was made but no fluid was evacuated. Two punctures of right temporosphenoidal lobe did not reveal any pus. One exploratory puncture of the cerebellum behind the sinus also failed to locate an abscess.

*January 9:* (Next morning.) Patient rational. Whole general condition apparently much improved. Temperature, 99.4° to 97.4° F.; pulse, 100-78. Later in day very restless, talking most of the time. Continued frequent small urinations, with involuntary bowel movements.

*January 10:* Patient still very restless, talks rationally at times, and then again rather hysterically. Complains of having no feeling in hands or feet. Uranalysis: acid; sp. gr. 1028; albumin, negative; sugar, negative; no mercurochrome.

*January 11:* Sudden paralysis of left side during night. (Hemiplegia.) Papilledema of both discs. Lumbar puncture: fluid 30 cc., turbid; 800 cells; no micro-organisms in smear. Three negative punctures of middle fossa through tegmen.

*January 13:* Temperature has been continuously high, 103° to 105° F. for past two days. Third severe chill. Second blood culture, negative.

*January 15:* Complains of severe pain in back. Lumbar puncture: fluid yellowish; blood present; coliform bacilli, same as found on swab from brain. Third blood culture, negative. Blood transfusion.

*January 17:* Exodus (seventeenth day of illness).

*Autopsy:* (Dr. Arthur J. Eagleton.) *Cerebral venous sinuses,* superior longitudinal sinus, lateral sinus, and left petrosals contain post-mortem clot. *Meninges:* Over cortex exhibit no macroscopical evidence of meningitis or

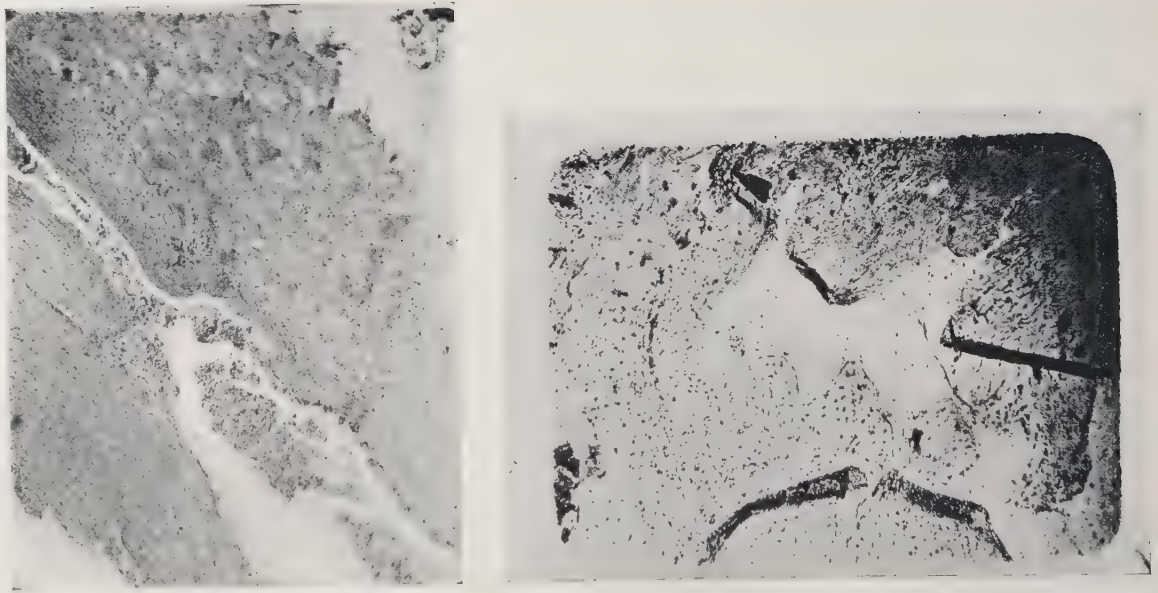
excess of fluid; but under the tentorium is a blood-stained collection of cerebrospinal fluid, such as had been removed by lumbar puncture.

*Brain* surface shows two areas of necrosis where the dura is adherent, one at the posterior part of the right temporosphenoidal lobe corresponding to the center of the area of the dura exposed by operation, another opposite the right internal auditory meatus, where a triangular zone of brain tissue is necrosed and covered by organizing purulent exudate. The latter area is sharply bounded internally by the basilar artery and occupies the cerebellopontine angle; the necrotic area being surrounded by a grayish discoloration of brain substance.

*Serial Sections of the Hardened Brain:* The lateral ventricle of the right side and its anterior superior horn are full of ante-mortem blood clot. There is no abscess, but the brain contains two areas of softening: (1) A zone in the hemorrhage itself 4 by 4 cm. in size, the center of which is situated in the parietal lobe 9 cm. from the occipital pole. Anteriorly, the abscess softening is in the temporosphenoidal lobe, 3 cm. from the circle of Willis. The softening does not communicate with the ventricle posteriorly, but traced forward does so; and the necrotic material in it is directly continuous with the blood clot in the ventricle. This clot is firm and recent in front; behind the area of softening it is again firm as it passes into the posterior ventricular horn. (2) There is a small area of necrotic softening under the patch of necrosis on the surface of the temporosphenoidal lobe.

*Interpretation from Post-mortem:* From all appearances it seems that the patient had an infection spreading from the right internal ear to the two separate areas of meningitis; one by way of the eighth nerve to the pontine angle, and the other to the temporosphenoidal lobe by continuity of suppuration. The patch on the under surface of the cerebellum apparently remained localized, as far as can be judged by gross examination.

Case M.—Hemorrhage into Ventricle.



CASE M.—Microphotograph of Section through carotid artery at the site of operation. Ligated artery shows patent wall extending to the adventitia with effusion of blood between the latter and the media. Localized atrophy of the intima.

The other path of infection appears to have spread to the center of the parietal lobe, where an abscess was formed. This abscess at first did not communicate with the lateral ventricle, but owing to ulceration of one of the large branches of the middle cerebral, hemorrhage took place into the ventricle. As traced forward the abscess cavity came nearer and nearer to the lateral ventricle into which the hemorrhage burst anteriorly; the clot in the posterior part of the abscess cavity being hard and apparently of much older date than that in the anterior horn of the ventricle.

*Post-mortem:* Examination of the carotid artery removed from the neck wound demonstrated that where the ligature had been applied, the vessel itself was no longer occluded, the ligature having been absorbed.

*Abdominal Viscera:* Liver pale, cloudy, degenerative. Spleen, pultaceous and almost diffluent.

*Microscopic Sections:* Kidney sections show hemorrhagic nephritis. Brachial vein section where the mercurochrome had been injected showed no macroscopic sign of thrombosis. Serial microscopic sections of the carotid artery and jugular vein (extending from the lower part of the neck wound to bifurcation and up to entrance into the brain), show that the catgut ligature had been completely absorbed in the ten days between its application and the death of the patient; also that where the catgut ligature was applied to the artery, the vessel was no longer occluded. There was also a beginning dissection channel in the artery between the intima and the adventitia though there was no thrombosis of the artery or vein.

*Comment:* Was the hemorrhage into the brain due (1) to the extension of the sepsis into the blood vessels; (2) to the ligation of the carotid, or (3) to the mercurochrome? Probably partly to all three causes, for although the post-mortem leaves little doubt that the local sepsis (probably assisted by the exploratory punctures) played the largest part, still an abscess of the brain itself could not alone

account for so large a hemorrhage, as on examination the post-mortem reports of brain abscesses fail to reveal this complication.[10]

The ligation of the carotid was also a factor, as the operation in numerous instances has been followed by hemiplegia; but the latter is usually of embolic, not hemorrhagic origin.

It is also probable that the injection of mercurochrome may have a toxic influence on the blood vessels as suggested by the reported case of "rupture of the aorta."[11]

## AFTER-TREATMENT OF ALL BLOOD STREAM INFECTIONS

In the future treatment of infective thrombophlebitis more and more attention will be paid to serotherapy and vaccinotherapy; for while no specific serum or vaccine has as yet been evolved for the usual pyogenic infections of the blood stream, the nonspecific "protein reaction" undoubtedly exercised a very favorable influence on several of the cases herein reported (Cases 12 and 23). With the idea of adding specificity to the usual blood transfusion (Cases 6, 17 and 25), I have, during past years, instituted a course of immunized transfusions immediately following the operation (Case 23), a procedure which I believe has had a marked influence in favorably combatting septic meningitis.[12]

[10] Eagleton, W. P.: *Brain Abscess,* Appendix II, *Cerebellar Abscess:* Analysis of pathological condition in reported autopsies of 125 cases, pp. 220-243.

[11] Loudon, J., of Toronto—Case 22, referred to by Young, H. H.; Hill, J. H., and Scott, W. W.: "The Treatment of Infections and Infectious Diseases with Mercurochrome-220 Soluble," *Arch. Surg.,* Vol. 10, pp. 813-924, May, 1925.

[12] Eagleton, W. P.: "The Surgical Treatment of Meningitis (Third Communication)," *J. A. M. A.,* December 13, 1924, Vol. 83, pp. 1900-1906.

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# IMMUNIZATION OF DONORS

BY

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When possible a professional donor is preferred. These are injected from time to time (about once a month) with a minimal reactive dose of a stock vaccine made from strains of the streptococci, pneumococci, staphylococci, and other organisms locally prevalent in suppurative infections of the throat, ear and nose.

This vaccine is prepared especially so that the response to it is wide and the dosage high. Donors treated in this way show in their blood agglutinins, and what is important, bacteriolysins. The agglutination, of course, is no measure of immunity; the bacteriolysis is more difficult to demonstrate but is a measure of immunity. This is the usual primary stimulus.

*Technic:* On the day preceding the transfusion or at least two hours before the operation, such donors receive a secondary stimulus. This is, if possible, a specific autogenous vaccine, derived from the case, prepared without heat or destructive chemical agents. If there is no time for this, then a dose of the stock vaccine is given. This secondary stimulus is a large single dose given intramuscularly on the preceding day or intravenously the same day.

If the professional donor is not employed, the secondary stimulus only is given.

The resulting immunity is much less than in the professional donor, but that it exists is proved by the work of Sir A. Wright and others, and is based on the theoretical supposition that in most normal adults a general basal immunity must have been established so that they are prepared to respond to the secondary stimulus. In some cases such an injection will be ineffective.

I have found that within three hours of the intravenous secondary stimulus the non-professional donor experiences chilly sensations; the professional rarely so.

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